

THE CLINICAL SIGNIFICANCE OF
CENTRAL SCOTOMATA
OF THE VISUAL FIELD.

Thesis submitted for the Degree
of Doctor of Medicine

by

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THE CLINICAL * SIGNIFICANCE OF
CENTRAL SCOTOMATA
OF THE VISUAL FIELD.

INTRODUCTION.

When the eye steadily regards a small object as a fixation point, not only that object but others in a certain area of space around it can be seen at the same time.

This area of space is termed the field of vision.

Certain conditions of disease giving rise to impairment of vision show themselves as alterations in the field of vision. These alterations are of two main types, contractions and scotomata.

SCOTOMATA.

A scotoma is an area of depressed vision inside the field and, strictly speaking, should be entirely surrounded by less depressed or normal vision. It may, however, connect by a channel with the periphery or the blind spot. A brief description of scotomata in general will prove helpful, and this

may be carried out under certain headings (modified from Traquair).

1. Position.

(a) Central (pericentral):

A central scotoma, accurately speaking, is one which involves the fixation area. As scotomata of the central area are so important, it is preferable to use the word pericentral for those which involve the fixation area and a more or less regular amount of field round about it.

(b) Paracentral:

Paracentral scotomata are those which involve a portion of the field near the fixation area and may or may not include it. According to their position they are called supracentral, infracentral, nasocentral and temporocentral.

(c) Centrocaecal:

The scotoma which lies between the fixation area and blind spot, and which may overlap the former and run into the latter, is called a centro-caecal scotoma.

2. Shape.

The shape of a scotoma depends upon the arrangement of the damaged nerve elements in the retina and may be -

- (a) Round or irregular, e.g. central and paracentral.
- (b) Oval, e.g. centro-caecal.
- (c) Hemianopic - always central and corresponds to hemianopic changes in the entire field.

3. Size.

Depends entirely on the number of nerve elements involved and in certain parts of the visual path a small lesion may produce a large defect.

4. The Intensity.

The intensity of a scotoma may vary from an absolute defect, in which there is no perception of light, to one so slight that it is almost impossible to determine it.

As regards central vision, the presence of 6/6 vision recorded by Snellen's types is no criterion that no central scotoma exists. It is a fact that considerable depression of central vision can be present before the vision falls below 6/6 level.

In addition, the intensity of the defect may not be the same over all the scotoma. There are often areas within the scotoma which are denser, forming as it were nuclei to the defect.

5. Margins.

The change between the scotoma and the rest of the field may be sudden or gradual. It may differ

VISUAL NERVE PATH

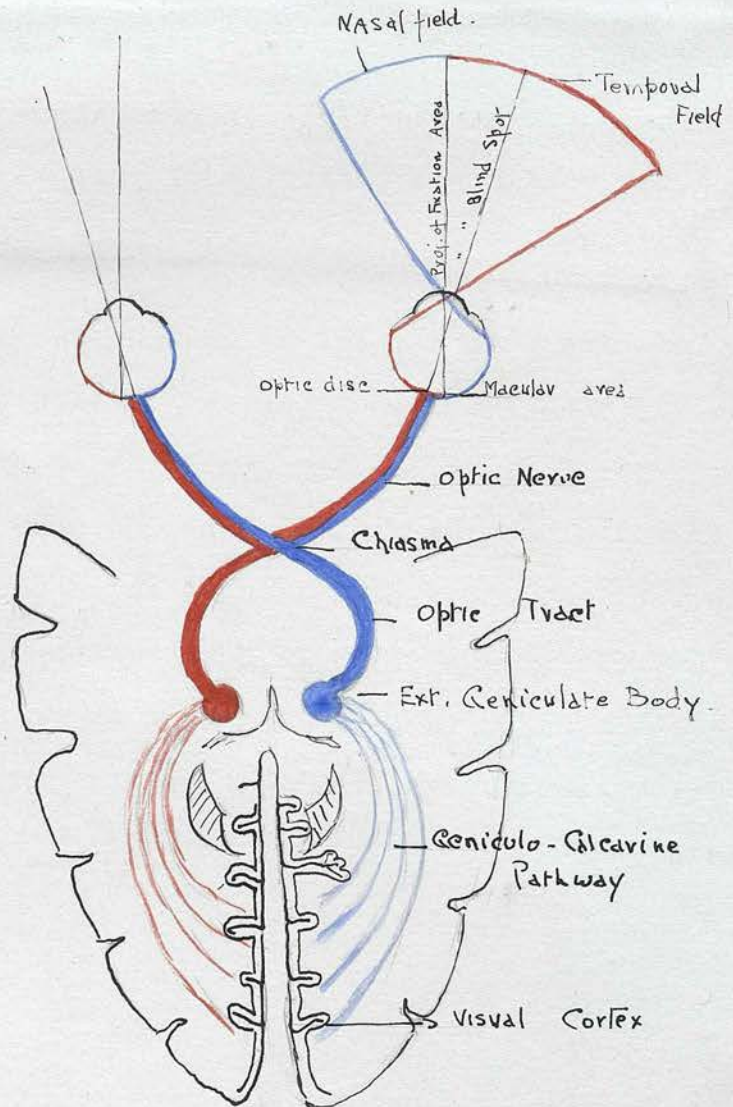


Diagram of Visual Nerve Path.

at various points on the margin and at different times.

6. Course.

The onset may be rapid or gradual, and the condition may remain stationary, or it may alter from time to time.

The term Central Scotoma must be taken to include the true central or pericentral scotoma, the paracentral scotoma and the centro-caecal scotoma, and it would be well at this point to consider the anatomy of the parts, a lesion of which produces such a field defect. It is most suitable to commence with the retina and travel in an afferent direction along the visual path to the occipital cortex.

ANATOMY.

STRUCTURE OF RETINA.

The retina is made up of a large number of layers and these consist, from without inwards, of the following parts:-

1. Pigment Epithelium -

This consists of a layer of polygonal pigment cells lying next the choroid. These cells have

GENERAL STRUCTURE OF THE RETINA

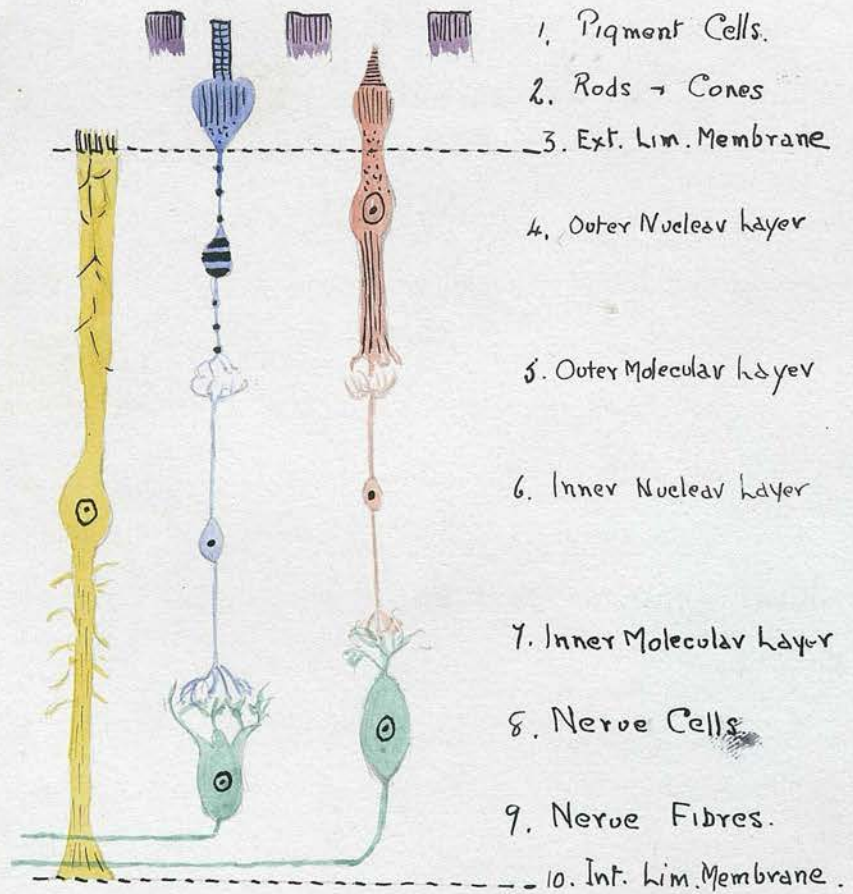


Diagram of General Structure of Retina
modified from Kyle & de Souza.

processes which dip down between the rods and cones of the next layer. The cells also secrete the visual purple or rhodopsin which is present in the rods.

2. Rods and Cones, or specialised nerve epithelium.

The rods and cones are the visual elements of the retina; the former are concerned with vision in low luminosities, while the latter are concerned with vision in bright luminosities. The rods contain visual purple, but there is no visual purple in the cones. The bases of both rods and cones perforate the external limiting membrane and are continued as rod and cone fibres.

3. The external limiting membrane is made up of the outer expanded portion of the supporting fibres of Muller.

4. The external nuclear layer consists of the fibre portion of the rods and cones with their nuclei and fine fibres belonging to the supporting cells of Muller.

5. The outer molecular layer contains a number of synapses between rod fibres and rod bipolar cells and between cone fibres and cone bipolar cells. It also contains a few neuroglial stellate cells.

6. The inner nuclear layer consists chiefly of rod and cone nucleated bipolar cells, which form the

first neurone on the visual path.

These bipolar cells send one process to the outer molecular layer to form synapses with rod and cone fibres, and another to the inner molecular layer to form synapses with the peripheral processes of the ganglion cells.

In this layer there are in addition the supporting cells of Muller and several others.

7. The inner molecular layer contains synapses between rod and cone bipolars and ganglion cells and many neuroglial cells.

8. Layer of ganglion cells.

These are large flask-shaped cells with a single process running inwards and several dendrites running outwards to form synapses with the rod and cone bipolar cells. They form the second set of neurones.

9. Layer of non-medullated fibres.

These are derived from the ganglion cells. They are afferent in function and gather together to form the optic nerve.

10. Internal limiting membrane is formed by the expanding bases of the supporting fibres of Muller.

Modifications of retinal structure.

In the periphery of the retina the rods and rod fibres are most numerous. As before stated, they are adapted for vision in low luminosities, which explains the fact that man sees better with the

peripheral part of the retina at night. A large number of these rod fibres are connected by means of the rod bipolar cells with one ganglion cell.

In the middle portion of the retina there are about an equal number of rods and cones and fewer of these are connected with one ganglion cell.

A very different state of affairs is present at the macula lutea or yellow spot. This is the most sensitive region of the retina. It is an elliptical area measuring about 2 mm. across and has a depressed centre called the fovea centralis.

At the macula the specialised nerve epithelium consists of long slender cones and their fibres, but there may also be a few rods present.

At the actual fovea the retinal layers are very thin and spread out. There are no rods present. The cones are very long and thin and each is connected with one ganglion cell. The nerve cells and fibres are displaced to the surrounding macula, causing the retina at this part to be slightly thickened, and here the sloping ganglion cells are several layers deep.

A lesion, even if very small, affecting this part of the retina depresses central vision very greatly and produces a central scotoma.

The nerve fibres are gathered together and leave the eye slightly to the nasal side of the

macula by the optic nerve. At this point the nerve fibre layer alone is represented and therefore a blind spot is produced in the field of vision.

The nerve fibres which come from the macular area in the retina are short and less curved than those lying near the horizontal meridian. They pass practically straight to the temporal side of the optic papilla.

At the optic papilla the nerve fibres are gathered together to leave the eye as the optic nerve.

The nerve fibre layer is particularly thick on the nasal side owing to the accumulation of fibres, and the temporal side is left free for the papillo-macular bundle which displaces the temporal fibres upwards and downwards.

COURSE OF PAPILLOMACULAR BUNDLE.

The arrangement of the fibres in the nerve and onwards is not precisely known as yet, but present knowledge is based on the work of Henschen and Roenne.

The macular fibres come from the central portion of the four retinal quadrants and are collected together to form a sector shaped area which lies in the outer part of the papilla and forms a fourth of it.

Immediately behind the eye the central part of the sector is formed of dorsal and ventral crossed macular fibres, while above and below the central part lie the dorsal and ventral uncrossed macular fibres.

The macular fibres then move towards the centre of the nerve and become surrounded on the outer aspect by the dorsal and ventral uncrossed peripheral bundles. At this same time the dorsal and ventral uncrossed macular fibres form into one bundle which lies to the lateral side of the bundle of crossed macular fibres.

In the posterior part of the orbital optic nerve the macular fibres form a central core.

Just in front of the chiasma the crossed macular fibres separate from the uncrossed. The crossed macular fibres lie dorsally in the chiasma and cross in the dorsal layers of its middle and posterior thirds. The posterior edge of the chiasma consists entirely of crossing macular area fibres.

The uncrossed macular fibres lie in the central part of the lateral halves of the chiasma.

In the tract the macular bundle is central in position. It lies closely along the connection of the tract to the brain, i.e. it is first central and dorso-mesial, then dorso-central, and lastly dorso-lateral and peripheral, and here it enters the geniculate body.

In the geniculate body the fibres lie in the dorsal layer of the middle and posterior parts of the ganglion and its posterior extremity.

In the optic radiation the macular bundle lies between the dorsal and ventral peripheral fibres.

The fibres of the macular bundle end in the most posterior part of the occipital centre, and the foveal fibres at the tip of the occipital pole.

INTERPRETATION OF FIELD DEFECTS FROM ANATOMICAL POINT OF VIEW.

In every case of a lesion of the visual path from the cells of the outer layers of the retina to the occipital cortex, a resulting defect in the field of vision must take place.

By a knowledge of anatomy of the part, a lesion of which gives rise to a certain type of field defect, we are able to diagnose the site of the lesion with considerable accuracy.

Retina.

Field defects which are due to lesions of the retina depend upon involvement of :-

- (a) The outer layers of percipient cells or photo-chemical apparatus, or
- (b) The inner layers of ganglion cells and nerve fibres or conducting apparatus.

(a) When the lesion affects the percipient cells, the field defect corresponds in position, size, shape and intensity to the lesion. For example, a small round patch of central choroiditis affecting the macular area would give rise to a small round central scotoma of intensity varying according to the severity of the lesion. In lesions of the photochemical apparatus, it is common to find a reduction in the perception of blue as compared to red. When the lesion affects the ganglion cells, there is relative red green blindness present and reduction of the perception of white. The defects are similar to scotomata due to lesions in the optic nerve, but differ from these because nerve fibre bundle defects are never present.

Note: Among the usual forms of defect due to interference with ganglion cells are centro-caecal, and round or irregular central scotomata.

(b) Defects produced by lesions of the nerve fibre layer correspond to the area from which the affected fibres come.

A small lesion in certain situations may therefore produce an extensive field defect.

The Optic Nerve.

The cross section of the optic nerve corresponds to the retina, therefore a lesion in the optic nerve

will cause a defect in the inverse portion of the field: e.g. a lesion in the lateral half of the nerve will cause a defect in the nasal field, while a peripheral or central lesion will cause a peripheral or central defect respectively.

Immediately behind the eyeball, however, a peripheral lesion would cause a defect of the central field because at this level the macular bundle of fibres lies to the outer side of the nerve and at the periphery.

At the chiasmal end of the nerve the crossed macular fibres separate from the uncrossed, giving rise to a possibility of involvement of one bundle without the other and therefore the production of unilateral hemianopic defects. These scotomata have been given the name of "junction scotomata" (Traquair) on account of their origin at the junction of the nerve and chiasma. The centro-caecal scotoma is very common as a result of interference with the optic nerve, but it does not appear to be related to any special part of the nerve.

The Chiasma.

Lesions in the chiasma produce bitemporal hemianopic defects. If the lesion is situated in or near the posterior part of the chiasma, a bitemporal hemianopic central scotoma is produced, owing to

interference with the macular fibres, which, as has already been seen, lie in the posterior part of the chiasma.

The Tract.

The typical defect produced by a lesion in the tract is a homonymous hemianopia, which may be congruous or incongruous.

In such a lesion the macular fibres may or may not be affected with corresponding presence or absence of central field defect.

Suprageniculate Pathway.

The typical defect produced by a lesion of this part of the visual path is a congruous hemianopia with sparing of the fixation area.

INTERPRETATION OF FIELD DEFECTS FROM PATHOLOGICAL POINT OF VIEW.

A. GENERAL.

On broad principles the more severe the lesion, the denser will be the defect. As all pathological processes vary in severity, and as they are surrounded by an area which is less severely

affected though not normal, it follows that there will be variations in the density of the field defect usually the centre being more dense and surrounded by an area which is less dense. If the pathological process is very acute, then the onset and increase in density of the scotoma will be rapid and vice versa. The same applies to resolution. The end result depends more often on the duration than the severity of the path^{ological} process. Even badly damaged nerve elements recover in a surprising fashion and only the most severely injured are permanently impaired. It is not possible to know from the field changes whether nerve cells are killed or only temporarily impaired from conducting visual impulses.

Disproportion between colour loss and white loss indicate a progressive lesion and bad prognosis, while if the fields for white and colour are proportionate, the lesion may be regarded as stationary.

Steeply sloping margins to a scotoma indicate a circumscribed area of affection, while a gradual slope indicates a diffuse lesion.

B. LOCAL.

1. Retina.

(a) Photo chemical apparatus.

The visual neurones are very sensitive to

alterations in nutrition. In the retina this may be caused by foci of inflammation in the choroid or defects in the circulation (retinal and choroidal), the presence of endotoxins or excessive light, damage by tumour growth and primary degeneration.

Field changes here are exaggerated in dim light and show deficiency in recognition of blue. Obviously they will produce a central scotoma only if the lesion occurs in the macular area.

(b) Conduction apparatus - i.e. ganglion cells.

The ganglion cells seem particularly susceptible to toxins and the field change consists of loss of perception for red and green, and to a less extent white. These cells are also considerably influenced by vascular changes.

2. Optic Nerve.

The optic nerve is very susceptible to toxins, both endogenous and exogenous, and to inflammation. Here also come into play such causes as pressure and vascular changes - but these are rarer. Injury, however, is common.

3. Chiasma.

Passing back to the chiasma, toxic and inflammatory changes are rarer, so also is injury, but pressure becomes a common and important factor, and

vascular lesions are not unknown.

4. Tract.

The tract is affected by similar conditions to the chiasma.

5. Suprageniculate Pathway.

Here the most common form of pathological lesion is vascular. Pressure comes second, but is much rarer, while toxic and inflammatory conditions and injuries (apart from war) practically do not occur.

GROUP I.RETINA AND CHOROID.

Obviously it is only when lesions affect the central part of the choroid and retina that the changes shown in the field will partake of the nature of central scotomata.

Perimetry is not of primary importance in many of these cases, but in those which are obscure or in which the ophthalmoscopic findings do not correspond to the severity of visual loss, it is often helpful.

OUTER LAYERS OF RETINA.(a) Macular choroido-retinitis.

In cases of central or macular choroido-retinitis, the diagnosis is usually made from ophthalmoscopic appearances, and the scotomata correspond more or less in extent and shape to the visible

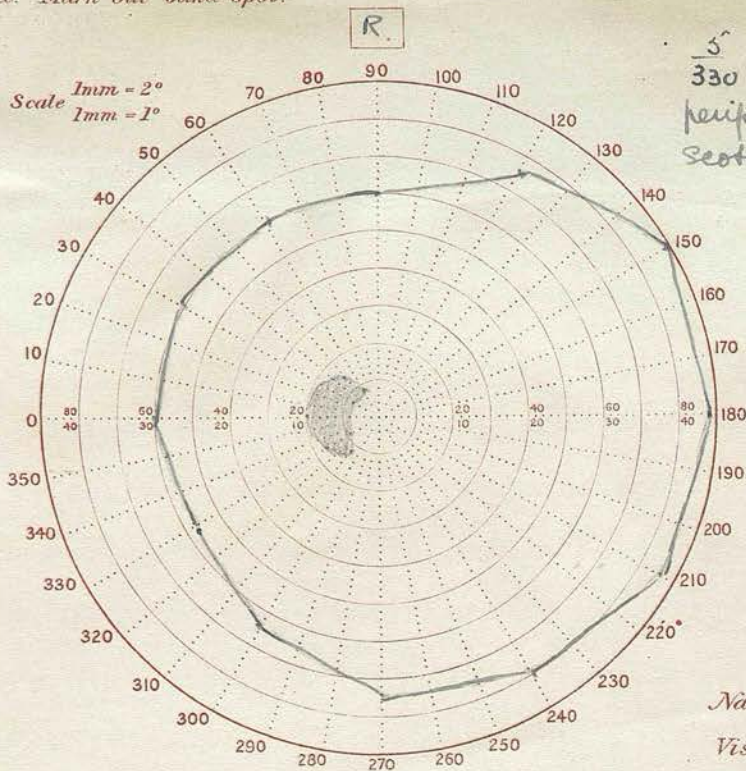
lesion.

In the early stages, when the condition is acute, the depreciation of vision is considerable and general.

The central part of the fundus is covered with a large number of small yellow foci of inflammation and in addition to this there is a larger surrounding area of retinal oedema which appears as a whitish slightly raised area. As the condition subsides, spots of pigmentation make their appearance and in the last stages the pigment is scattered all over the affected area in typical bone corpuscle patches. It is often very difficult to judge the amount of vision present from the ophthalmoscopic appearances, as apparently severely damaged cases often have surprisingly good vision, and vice versa.

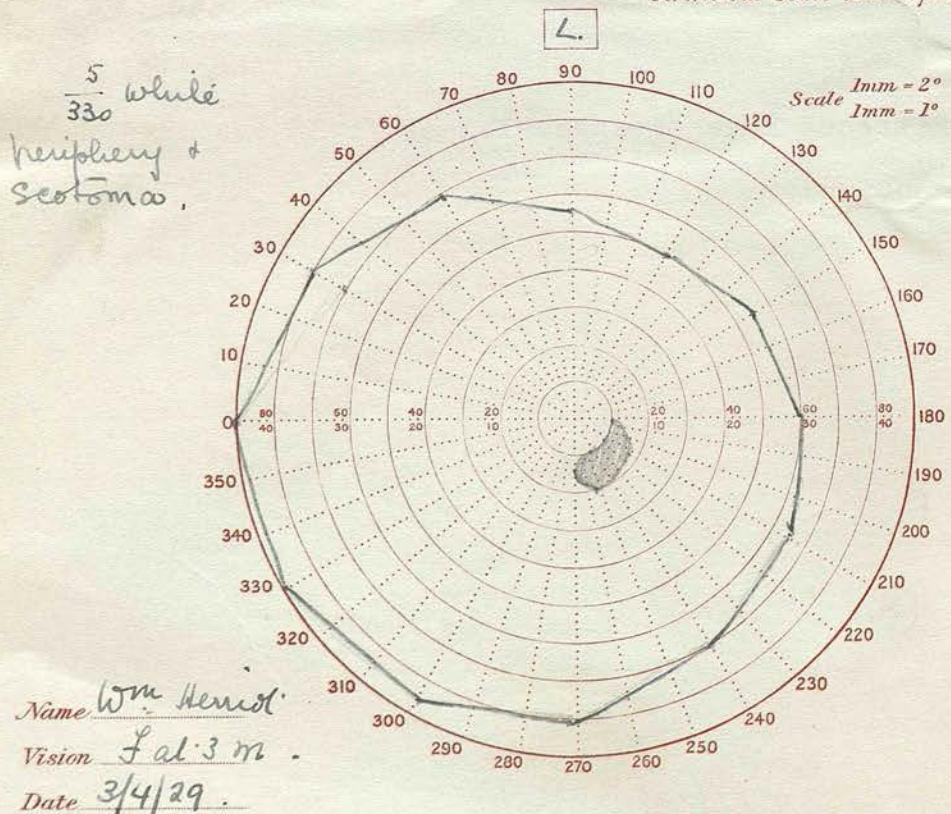
Two illustrative cases of macular choroido-retinitis follow - one severe, of which the cause is syphilis, and the other slight, of which the cause is unknown.

d. Mark out blind spot.



WM. HERRIOT

Stroke out Scale not requir



Case I. Case of Paracentral Scotoma from Central Syphilitis Choroido-Retinitis.

2.4.29. William Herriot. Age 34.

Complaint: Blurred vision in both eyes.

History: Nine days previously, sight of left eye suddenly became blurred. Two days previously, sight of right eye went the same way.

Examination: Pupil reactions normal.

Tension normal. Refractive error negligible.

Vision of both eyes counting fingers at about 3 metres.

Central area in right fundus appears pale and in left fundus pigmented.

Discs appear normal.

Widespread active condition of choroiditis and vitreous haze made results of field examination inconclusive.

3.4.29. Field taken with $\frac{10}{330}$ and $\frac{5}{330}$ white periphery normal - paracentral scotoma for $\frac{5}{330}$ white in each eye as an illustration of correspondence between lesion and field defect. There was no sign of even a minute ring scotoma.

4.4.29. Pupils dilated.

Central area of right fundus shows oedema of retina.

Central area of left fundus shows many spots of

central choroido-retinitis with commencing pigmentation. Atropine ordered.

R.V. = F. at 3 M. L.V. = 6/80.

5.4.29. I.S.Q. as regards fundus picture.

R V. = 6/80. L.V. = 6/36.

9.4.29. R.V. = 6/36. (Pigmentation now commencing.) L.V. = 6/18.

12.4.29. R.V. = 6/18. Pigmentation. L.V. = 6/18.

19.4.29. R.V. = 6/18. L.V. = 6/18.

23.4.29. R.V. = 6/18. Much pigmentation in R. fundus now. Wassermann reaction strongly positive. Referred to Venereal Diseases Department for anti-syphilitic treatment.

14.10.29. This case was seen again six months later and then showed the typical end picture of choroido-retinitis. The central area of each fundus was covered with a large number of spots of black pigment resembling bone corpuscles. The left fundus was considerably more pigmented than the right, and this was borne out by the record of vision.

R.V. = 6/9. J.1. L.V. = 6/18. J.3.

There was no sign of oedema and the active process was obviously at an end. Anti-syphilitic treatment was still being carried out.

Charts of the fields of vision were not taken

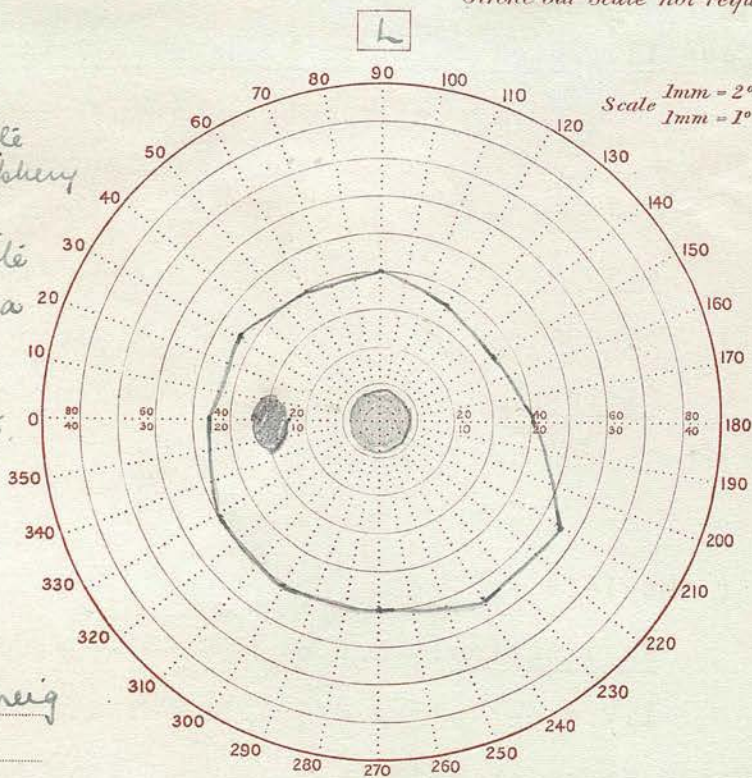
CASE II

CENTRAL CHOROIOIDO-RETINITIS

MRS GREIG

Stroke out Scale not require

$\frac{1}{330}$ white periphery
 $\frac{1}{2000}$ white scotoma
 $\frac{20}{2000}$ white blind spot



Name Mrs Greig

Vision 6/6

Date 9/5/29.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

on this occasion, as no useful information could be gained thereby.

19.5.30. Examined seven months later, condition unchanged.

Case II. Case of Central Choroido-Retinitis in One Eye. (Cause doubtful)

8.5.29. Mrs Greig. Age 34.

Complaint: "Blank spot in centre of vision of left eye only."

History: Patient stated that for one month previously she had been conscious of a small but definite round blurred area in the centre of the vision of the left eye.

Examination: R.V.c. - 1.75 = 6/6 J.1.

L.V.c. - 1.75 = 6/6 J.1. but dimmer than right. Field taken showed small central scotoma for 1/2000 white. After the pupil was dilated, a small patch of choroido-retinitis was seen at the macular area.

The patient was examined by a Physician and the following report given:-

History: Two years previously, right ovary and tube removed. Question of tuberculosis. One year ago, operation for duodenal ulcer. Cured twice for pelvic inflammation. Has had mucous colitis several times.

March, 1929. Influenza.

April, 1929. Dimness of vision in R. eye.

Present condition:

Blood: Red cells 4,900,000

White cells 10,000. On high side

but explainable by septic focus in teeth.

Hb. 82%. C I .83.

Wassermann negative.

Urine: Normal.

P.V. Normal. No venereal disease.

No signs of tuberculosis.

No septic focus found except in teeth.

Dentist's report:-

Several teeth were badly decayed and signs of pyorrhoea were present.

All bad teeth extracted - replaced by false.

November, 1929. Patient reported vision as not having improved.

In this case there are two points which strike one regarding the cause of the inflammation. One is the fact that one month before the sight of the right eye was affected, patient had an attack of influenza; and the other is that her teeth were very septic. Either of these conditions might cause the inflammation of the choroid. Being at the macular area, the lesion, though small, was immediately noticed through causing a central scotoma, due to interference with the cells of the outer

layers of the retina.

January, 1930. Complete re-examination -
Condition unchanged.

May, 1930. Complete re-examination - Condition
unchanged.

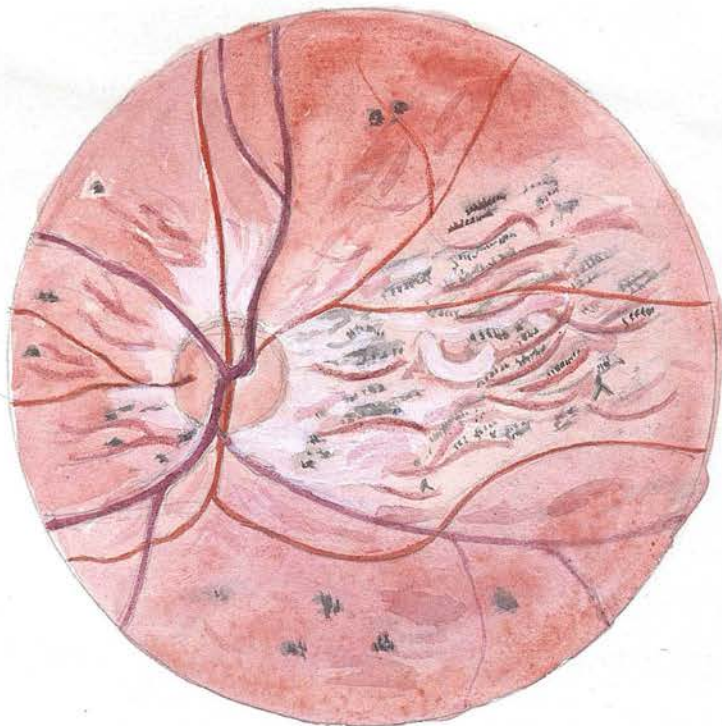
(b) Retinitis pigmentosa.

This disease, which is a degenerative change, is commonly hereditary and usually characterised by a ring scotoma corresponding to the situation of the affection in the area of the fundus between the macula and the extreme periphery. Central scotoma is rare.

The case of Alex. Hocking, which follows, appears to be of a similar type of disease affecting the central part of the fundus instead of the periphery.

The whole of the central part of the fundus was covered with pigmented spots and with white areas of atrophy, in addition to which there were a few typical bone corpuscle spots of pigmentation in the periphery.

Central vision was very poor, being only the counting of fingers at 4 or 5 metres. The patient had obviously a dense central scotoma, but this could not be charted owing to his inability to fix. The peripheral field was charted as well as possible

CENTRAL CHOROIDO-RETINAL ATROPHYALEX⁷ HOCKING

and showed normal limits for $5/330$ and $1/330$ white test objects.

Alex. Hocking. Age 56. Trader (S. Africa).

History:

In 1912, first noticed difficulty with reading.

In 1914, never saw well at a distance, but getting worse now.

In 1916, went to Johannesburg and saw specialist. Ordered glasses, but no use. Had various pairs of glasses; all no use.

In 1917, had blood test taken in Basutoland - 2 injections given.

Family History:

Father, very good. Two brothers.

Mother and her family all wore glasses, but no history of this complaint.

1. Sister, 61. Glasses for reading.

1 daughter (36), all right.

2. Sister. Glasses for all purposes.

Boy, 21, turned down for navy.

Girl, 24.

3. Patient. No family.

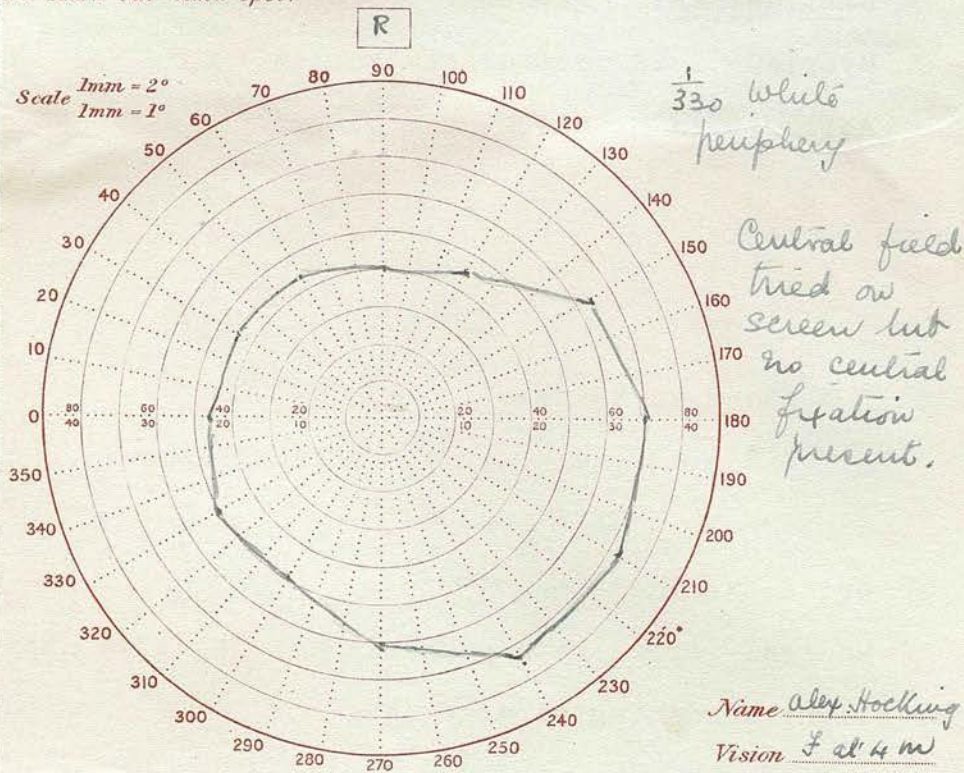
4. Brother, 54, farming; unable to read for a year. Worse than patient.

2 boys, 23 and 27, all right.

1 girl, 26, all right.

CASE OFCENTRAL CHOROIDO-RETINAL ATROPHYALEX. HOCKING

d. Mark out blind spot.



THE DARIEN PRESS, 5 BRISTOL PLACE, EDINBURGH.

5. Brother, 50. All right.

2 girls, 12 and 17, all right.

6. Sister, 46. Glasses for reading.

1 girl, 21, all right.

Examination: Pupillary reactions normal.

Myopia - 1.25 neutralises shadow.

Astigmatism with ophthalmometer.

R. O.

L. O.

R.V. c - $\frac{2}{-0.5}$ = Fat 5m.	L.V. c - $\frac{2}{0.5}$ = Fat 4m. 1
90°	90°

With + 3 added reads Cowell 12.

A large hand magnifier was no improvement.

Wassermann reaction negative.

Urine examination negative.

Medical report by a physician.- Negative except for slight hardening of arteries.

(c) Choroidal Changes in Myopia.

In very high degrees of myopia, changes in the eye grounds are common. There are large areas of atrophy alternating with black patches of pigmentation, and sometimes haemorrhages occur. The commonest parts of the fundus for affection are round the disc and in the macular area. If the changes take place in the macular area, there will be corresponding changes in the field of vision, and central and para-central defects occur which can be

HAEMORRAGE AT MACULA IN MYOPIA

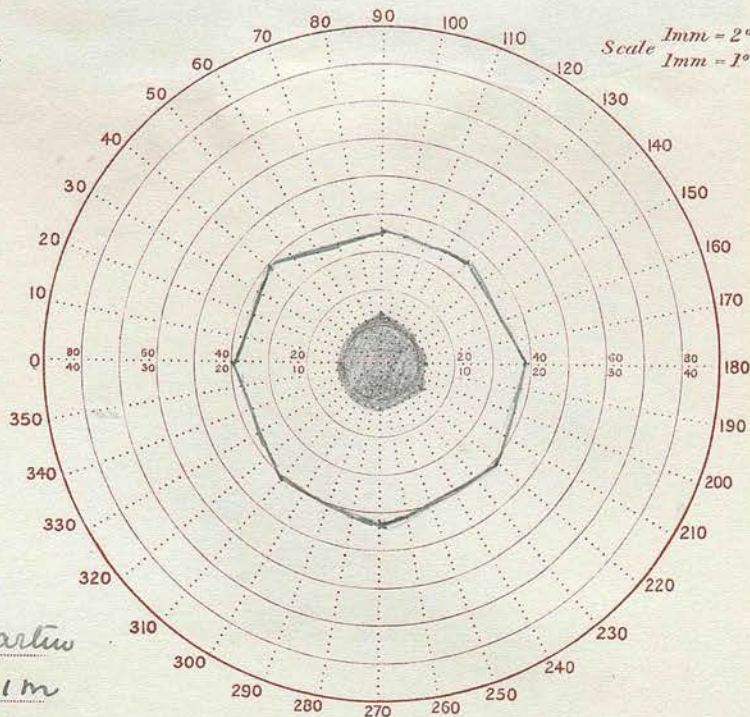
MRS MARTIN

Stroke out Scale not requ

L.

1. white
field &
scotoma

Scale Imm = 2°
Imm = 1°

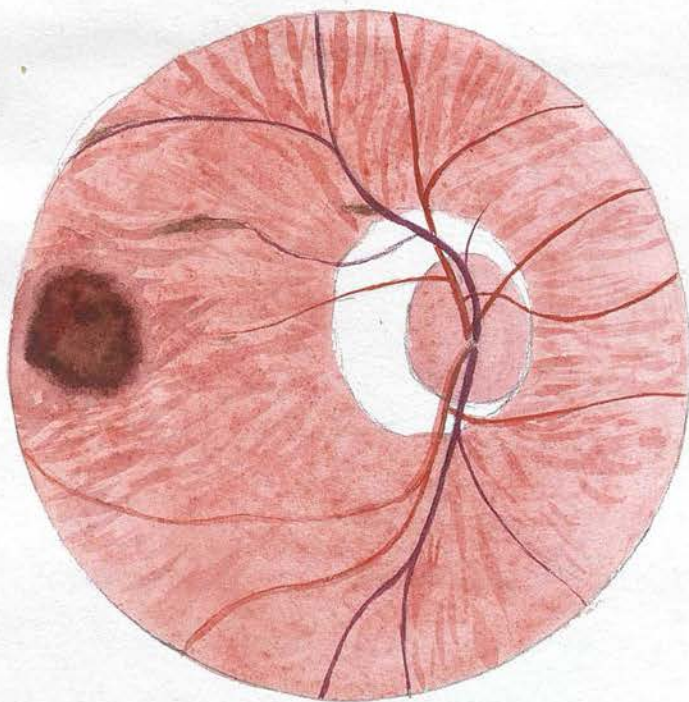


Name Mrs Martin

Vision 4 at 1m

Date 19.5.30.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

HAEMORRAGE AT MACULAMRS MARTIN

correlated with the ophthalmoscopic findings.

Case of Myopia with Macular Haemorrhage.

Mrs Martin, 6 Livingstone Place. Age 52.

Complaint: Black spot in front of left eye.

History: Patient gave a history of having suddenly discovered a black spot in front of the left eye one month previously. She had always been short-sighted.

Examination: 19.5.30.

R.V. \bar{c} - 5 - 6/9+ L.V. = Finger counting at 1
metre.

With naked eye J 1 No improvement.

Fundus of right eye was healthy except for slight choroidal atrophy round optic disc. In centre of left fundus a large reddish-brown area was present - obviously a haemorrhage. Chart of the field showed a central scotoma with a $\frac{1}{330}$ white.

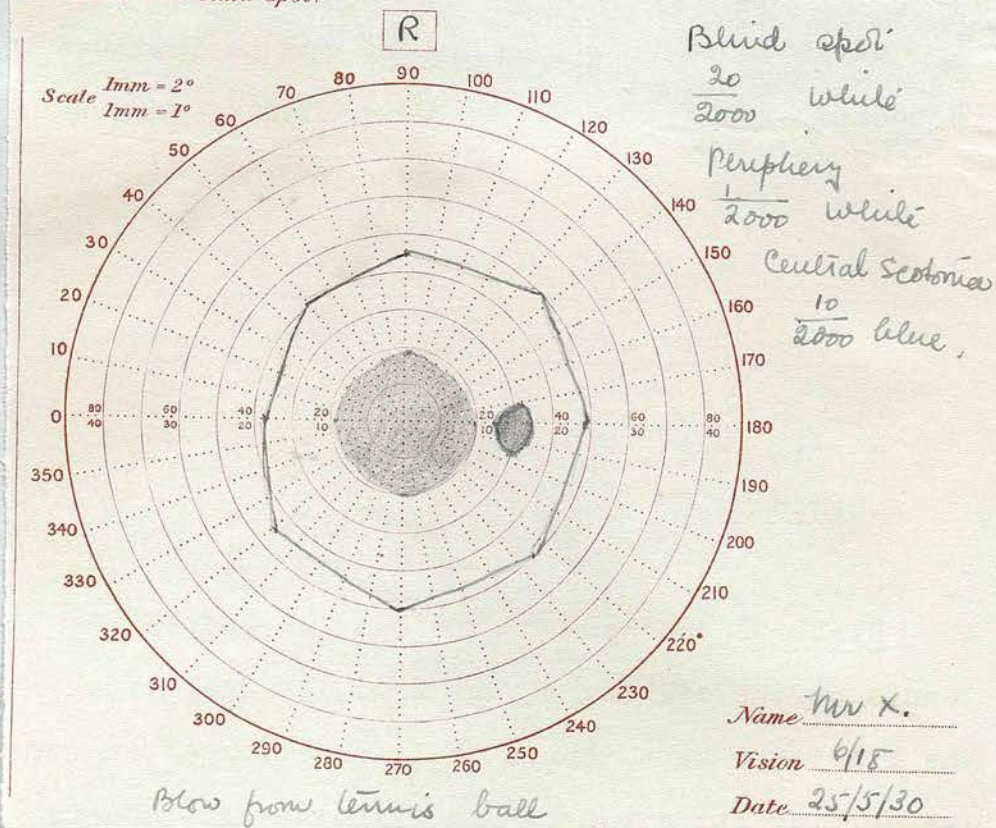
(d) Injury.

In cases of rupture of the choroid, there is no sign of a scotoma corresponding to the crescentic rupture in the choroid, showing that the retina traverses the rupture intact, but in cases in which there is oedema of the central part of the retina following a blow (commotio retinae), there is present a relative central scotoma for blue.

MACULAR HAEMORRAGE following INJURY

INDIAN STUDENT

red. Mark out blind spot.



In macular oedema from other causes, e.g. albuminuric neuro-retinitis, high intracranial pressure with swelling of optic disc, a similar central scotoma is often found.

Cases in which a blow on the eye is followed by haemorrhage at the macula give rise to central scotoma.

Case of Haemorrhage at Macula following blow from Tennis Ball.

Mr X., Indian Medical Student. Age 24.

25th May, 1930.

Complaint: Inability to see with right eye.

History: Patient stated that while playing tennis on the evening of May 23rd, he was struck on the right eye by a tennis ball, and thereafter could see nothing but a blur. In his own words, there was an appearance as of "green foliage" in front of the right eye.

Examination: Externally the eye was slightly congested and watering. Pupil reaction to light somewhat sluggish. Ophthalmoscopically, at the macula could be seen an area about the size of the disc, greyish red in colour and striate in appearance. This was obviously a haemorrhagic area.

Right vision = Less than 6/80. Left vision - 6/6.

May 27th. When re-examined on May 27th, vision had commenced to improve. Patient now stated that objects when looked at with right eye, seemed smaller and further away than with the left eye.

The haemorrhage was less dense.

R.V. = 6/18. L.V. = 6/6.

The field of vision showed a normal blind spot for 20/2000 white, normal periphery for 1/2000 white, with no central scotoma for 1/2000 white, but a pericentral scotoma extending roughly 9° all round was present for blue. Over this area blue was called white, but beyond it, was recognised as blue.

During the testing of the field, patient's eye became rapidly tired, and he had great difficulty with fixation.

(e) Eclipse Scotoma.

After every eclipse of the sun, a certain number of people present themselves for examination at an ophthalmic clinic with the complaint that they see a dark spot in the centre of the field of vision.

The history given is one of having looked at the sun without any protective glass, or with one insufficiently smoked.

Troublesome after-images are seen by everyone after looking at a sunset, but these are of a temporary nature only. Permanent disturbance results

from prolonged looking at the sun with insufficient protection.

An area of indistinct vision in the field remains (i.e. central scotoma) corresponding to that part of the retina upon which the image of the sun was cast upon the retina (macula lutea). Vision by Snellen's types is often good and ophthalmoscopic examination may be negative, but even in these slight cases, reading vision is impaired.

In severe cases, however, central vision is greatly depressed and on examining the fundus with the ophthalmoscope, pigmentary changes are seen at the macular area.

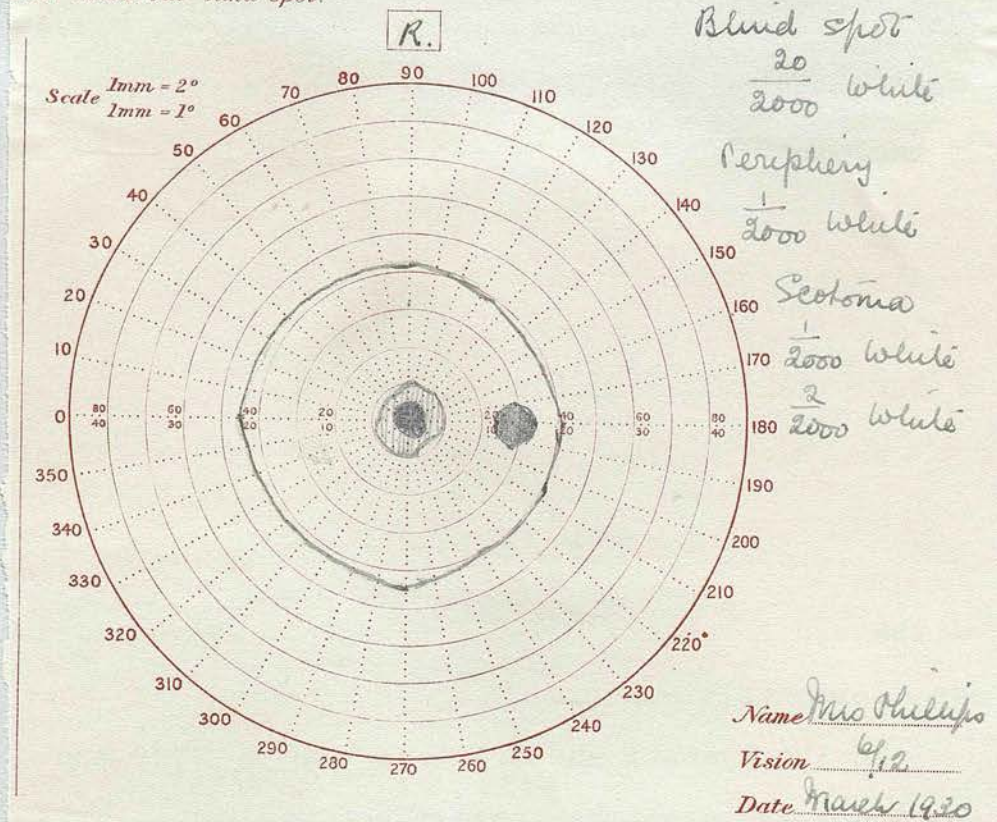
The scotoma is usually small and round and either exactly central or more commonly extending more to one side of the fixation point than another, it may be relative or absolute.

Inflammatory or degenerative changes are produced in the retina by the action of strong light, and to these changes are due the pigmented spots seen at the macula by the ophthalmoscope. The lesion is in the outer retinal layers. If the lesion is large and severe, the prognosis is bad, but with slighter cases, recovery is good. A certain amount of permanent impairment is always present.

The presence of a central scotoma in an old

CASE I ECLIPSE SCOTOMAMRS PHILLIPS

red. Mark out blind spot.



standing case of eclipse blindness indicates a certain amount of permanent destruction of the visual cells (outer layers) of the retina.

If the scotoma is small and relative, distant vision by Snellen's types may be unimpaired, while reading vision is only slightly interfered with. On the other hand, if the scotoma left is large and dense, there will be a good deal of loss of not only reading but distant central vision.

Case I. Eclipse Scotoma.

Mrs Blanche Phillips, 6 Brunton Terrace, Edinburgh. Age 54.

History: In 1911, patient looked at an eclipse of the sun through a hole in cardboard, with right eye, for nearly 2 hours.

In the evening she tried to read a paper and could not see even the largest print.

The condition improved steadily up to present date. Now she is not conscious of the spot when using binocular vision, but only when using right eye alone.

Examination: March, 1930.

R.V. = 6/12

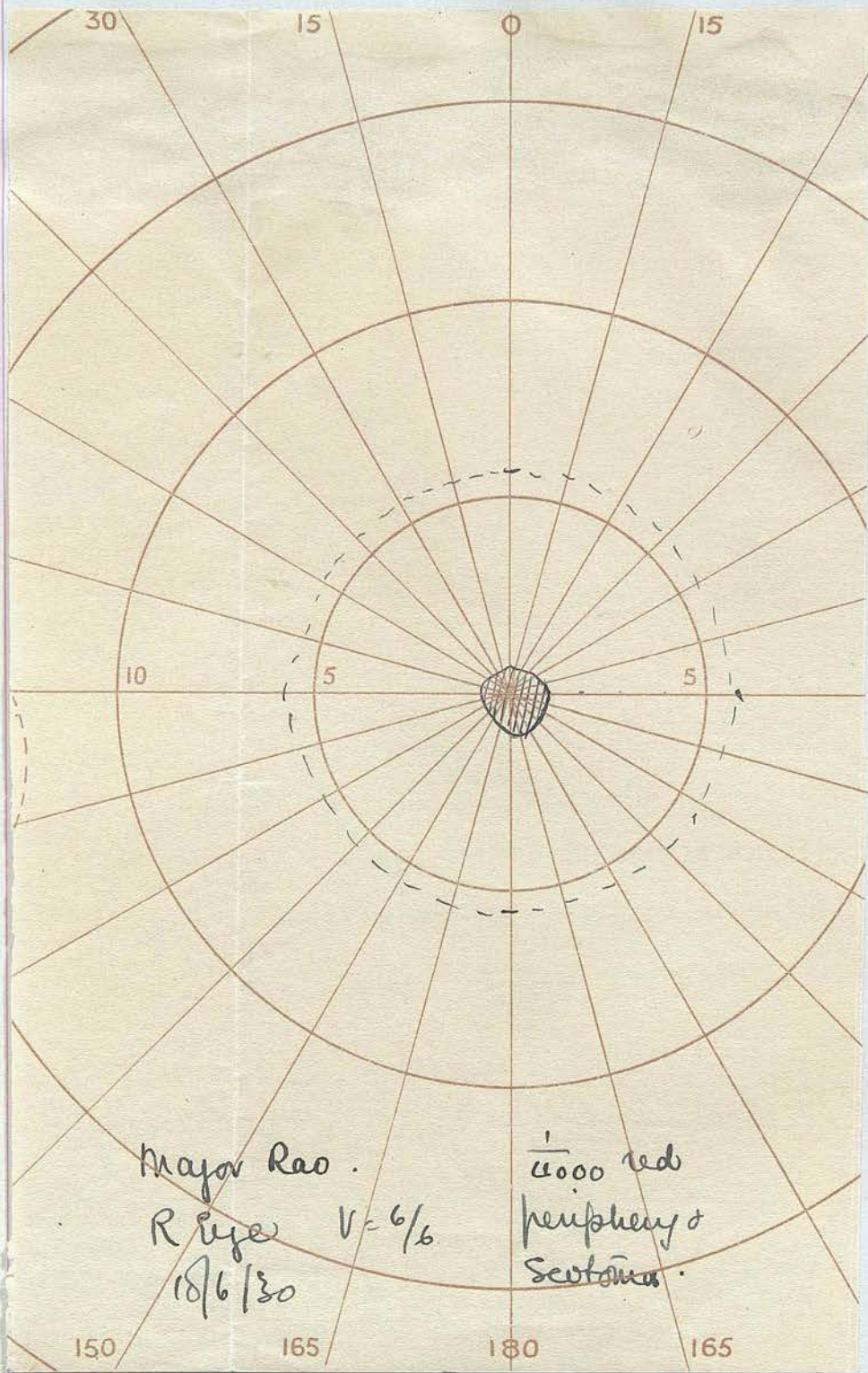
$\bar{c} + 2.5$ J 2.

L.V. $\bar{c} + 0.5$ = 6/6

$\bar{c} + 2.5$ 180°
 $+ 0.5$ = J 1.
 180°

There is a very small pigmented area present

MAJOR RAO



at the right macula. No other changes seen.

Small central scotomata are present for 1/2000 white and 2/2000 white.

Case II.

Major Rao, Indian Medical Service. Age 40.

18.6.30. History: Two years previously patient looked at eclipse of the sun with insufficient protection and received a slight burn of retina.

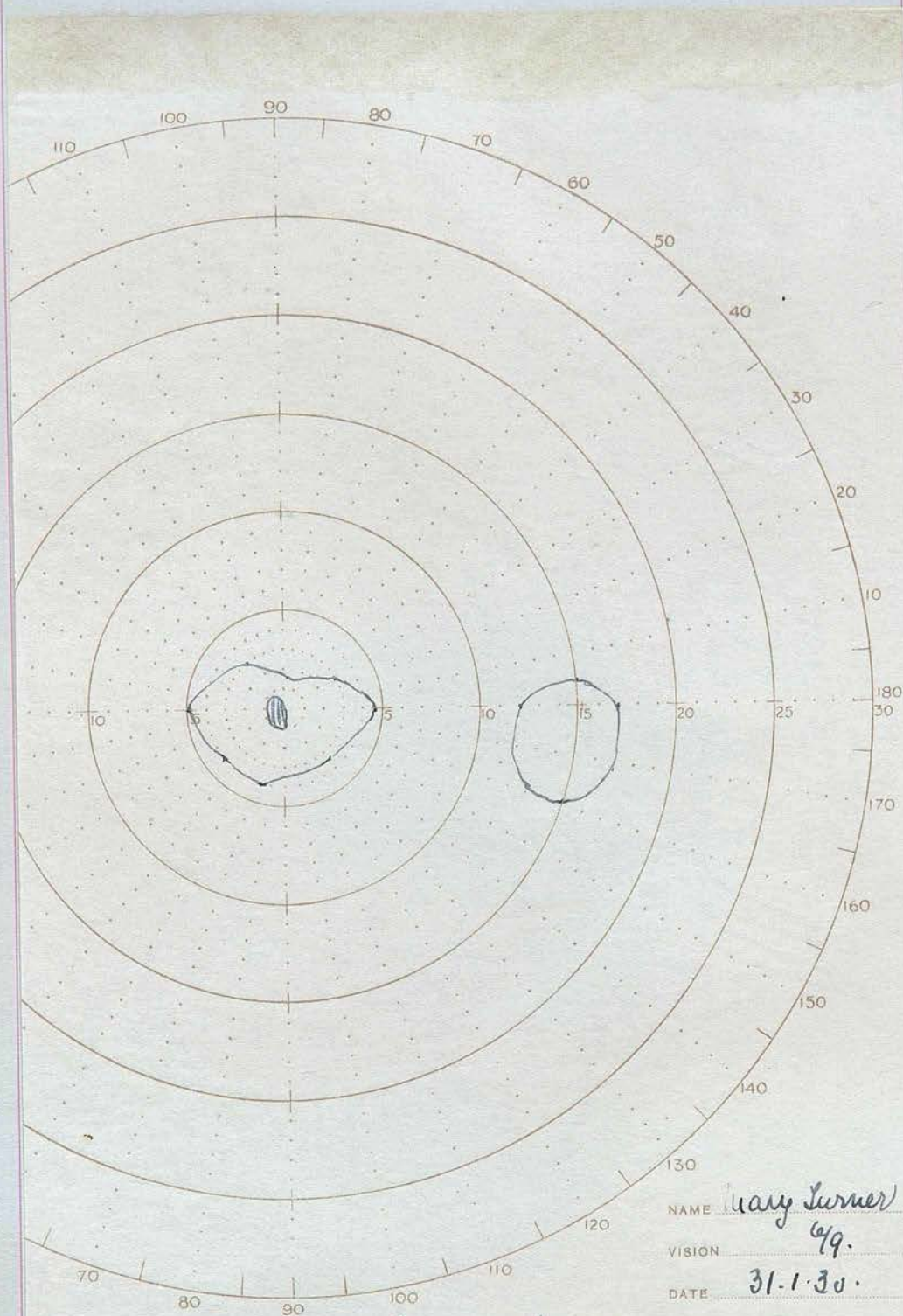
Examination: Pupillary reactions normal.

$$\begin{array}{rcl} \text{Right vision} & \bar{c} \begin{array}{l} - 1.5 \\ - 0.25 \end{array} & = 6/6 \\ & & 165^{\circ} \end{array}$$

$$\begin{array}{rcl} \text{Left vision} & \bar{c} \begin{array}{l} - 1.5 \\ - 0.25 \end{array} & = 6/6 \\ & & 180^{\circ} \end{array}$$

Slight pigmented lesion present at macula.

In order to bring out the presence of the central scotoma, it was necessary to test with a 1 mm. red test object at a distance of 4 metres. The periphery for this object was normal, and a small central scotoma about 2° diameter at its widest part was present.

MARY TURNER

NAME Mary Turner
VISION 99.
DATE 31.1.30.

Street, London, W.1.

$\frac{3}{2000}$ red Murphy & scotoma
 $\frac{10}{2000}$ white blind spot.

Case III. (By permission)*Case examined by RT — .*

Mary Turner, Servant, The Village, Edrom,
Berwickshire. Age 21.

30.1.30. Complaint: Headaches.

History: In 1920 looked at eclipse with insufficient protection and now has metamorphopsia in right eye.

Examination:

$$\text{R.V. } \bar{c} \begin{smallmatrix} +0.5 \\ +0.25 \end{smallmatrix} = 6/6 \\ 90^\circ$$

$$\text{L.V. } \bar{c} + 0.75 = 6/6$$

Lesion at fovea in right eye. Field changes.
Normal blind spot with $\frac{20}{2000}$ white. Normal periphery with $\frac{3}{2000}$ red. Small central scotoma with $\frac{3}{2000}$ red.

GROUP II.OPTIC NERVE.INTOXICATION AND INFLAMMATION.A. TOXIC AMBLYOPIA.

When considering the question of toxic amblyopia, the name is applied only to the result of poisons taken into the system, i.e. exogenous, although it must not be forgotten that certain systemic diseases, e.g. diabetes, also produce toxic amblyopia (endogenous). Of these toxins there are, according to Unthoff, two main groups:-

1. Those which produce peripheral neuritis and mainly give rise to central field changes, e.g. lead, alcohol.
2. Those which do not produce peripheral neuritis and are associated with peripheral field changes, e.g. quinine.

Tobacco amblyopia belongs to the first group as regards the field changes, but is an exception in that it does not produce peripheral neuritis.

TOBACCO AMBLYOPIA.

Tobacco amblyopia is by no means an uncommon condition, especially in the hospital class of patients.

The type of patient and the history which he gives often enable a diagnosis to be made practically before examination. The patient is usually a man of middle or old age who has a definite smell of stale tobacco hanging about him. He states that his vision has recently become poor both for reading and distance, and he sees people's faces blurred, as if he observed them through a fog. Enquiry elicits the fact that he smokes anything from 2-7 ozs. per week, usually strong black tobacco, and always a pipe.

Tobacco amblyopia from cigarette smoking is very rare, but chewing and the old fashioned snuff taking both cause it.

On objective examination, the eyes are usually quite healthy, but it is not uncommon to find pallor of the optic disc on the temporal side.

On subjective examination, vision may be reduced to any degree and the reading is poor. It is proportionately worse than one would expect from the distant vision.

Field Changes.

The scotoma of tobacco amblyopia is not a true pericentral scotoma. It lies between the blind spot and the fixation point and is therefore centro-caecal in position.

It is most easily demonstrated by a small red object and is always bilateral.

One eye is always further advanced than the other, which seems as if the condition was unilateral in its earliest stages.

A rough test which is useful as a preliminary to more minute investigation is carried out as follows. Cover the patient's left eye and get him to fix the right eye on a small white object. Hold a medium sized red object between the blind spot and the fixation spot and ask him its colour. The answer is usually as follows, "darkish", "brown", "pale", or "nothing". Next hold the test object to the nasal side of the fixation spot, when it will be immediately recognised as red. Repeat the test with the left eye. This will show similar findings.

The size of the red test object used must be varied to suit each individual case.

A more detailed examination discloses further features as follows.

In the earliest stages the scotoma lies between the fixation point and the blind spot and is

demonstrable only with a small red test object. At this stage the peripheral field is normal for $\frac{1}{2000}$ white. As time goes on the centrocaecal scotoma for red increases in size and may develop within itself one or two denser areas or nuclei. Of these one will lie to the nasal side of the blind spot and the other to the temporal side of the fixation area. The scotoma will then pass into the pericaecal amblyopia area on the one hand, and overlap the fixation area on the other. At this stage the patient's vision becomes poorer and if the peripheral field for $\frac{1}{2000}$ white is examined, it will show a defect on the temporal side. The defect for colour is always much greater than that for white.

As the patient gets worse, the defect increases in size and intensity until vision has deteriorated to counting of fingers at 2 or 3 metres distance.

The only treatment is to stop smoking. In some cases the vision not only shows no immediate improvement, but may actually get worse. This proves that tobacco is cumulative in its action.

In milder cases improvement usually sets in at once, and in all but the most severe, will occur sooner or later. During recovery the field defects retrace their steps. In a case where the scotoma has broken through on the temporal aspect, the colour field will creep round this side again. As

the scotoma recedes from the fixation area, vision improves suddenly. Usually the nuclei are the last to go. After two months' abstinence from smoking there is usually a great improvement in the vision.

Pathology.

From the field defect the lesion is obviously one of the conducting apparatus, but there is nothing to show whether it is in the inner layers of the retina or in the nerve itself. No other type of field change other than the above is ever produced in tobacco amblyopia.

The toxin of tobacco must have an affinity for certain nerve cells, although it is not yet known which. Macular fibres are never the first nor the most severely involved elements.

Clinical Significance.

From the clinical point of view, the typical centro-caecal scotoma of tobacco amblyopia is of enormous importance. When seeking a cause for the diminished vision, no abnormality in refraction media or fundi may be found. Hence the great importance of the field test.

This should be carried out on the Bjerrum screen. In nearly every case a $1/2000$ or $2/2000$ white will give the necessary peripheral field. In severe

cases the field for $1/2000$ may be contracted or fall inside the blind spot. In milder cases the field for $1/2000$ will be normal. The scotomata show with varying sizes of red and lie in the centro-caecal area. As the field defects in these cases have been found to be consistent, the diagnosis of tobacco amblyopia can be based on the perimetric findings. Diabetics who smoke are very prone to develop tobacco amblyopia, and it is more likely that the scotomata found in these cases are due to the tobacco rather than to the diabetes.

Tobacco amblyopia also develops in patients after a debilitating illness with rather more than usual rapidity and has been known to appear almost instantaneously after an accident or sudden fright.

There should be no difficulty in diagnosing tobacco amblyopia from other forms of retrobulbar neuritis if the field defects are carefully charted and studied, especially if the whole clinical picture is taken into consideration.

Tobacco amblyopia occurs in women who smoke a clay pipe.

ALCOHOL AMBLYOPIA.

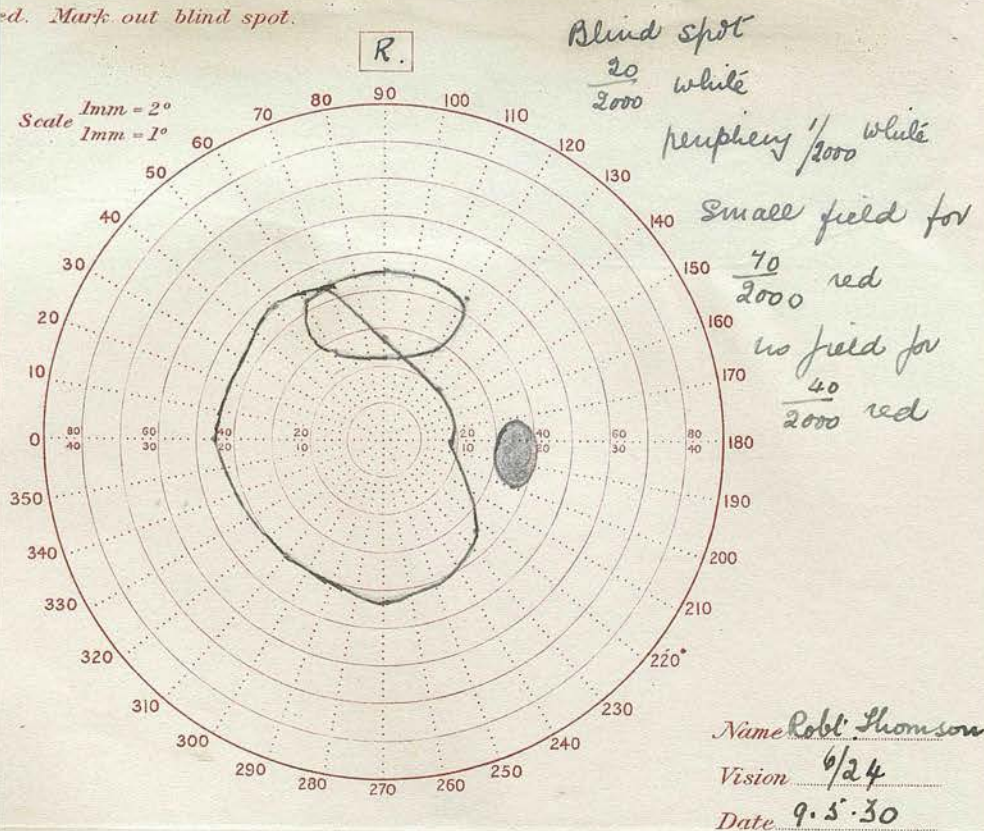
It is still a debatable point as to whether alcohol as such causes amblyopia. Many observers abroad are inclined to think that alcohol alone can

produce an alcohol amblyopia or form with tobacco a potent factor in the production of a "tobacco-alcohol" amblyopia. On the other hand, in this country, observers believe that pure alcohol does not either with or without tobacco produce an amblyopia, and any amblyopia which does occur as a result of alcohol is due to the impurities therein, e.g. fusel oil. The amblyopia which occurs in the form associated with tobacco is probably due to the tobacco alone.

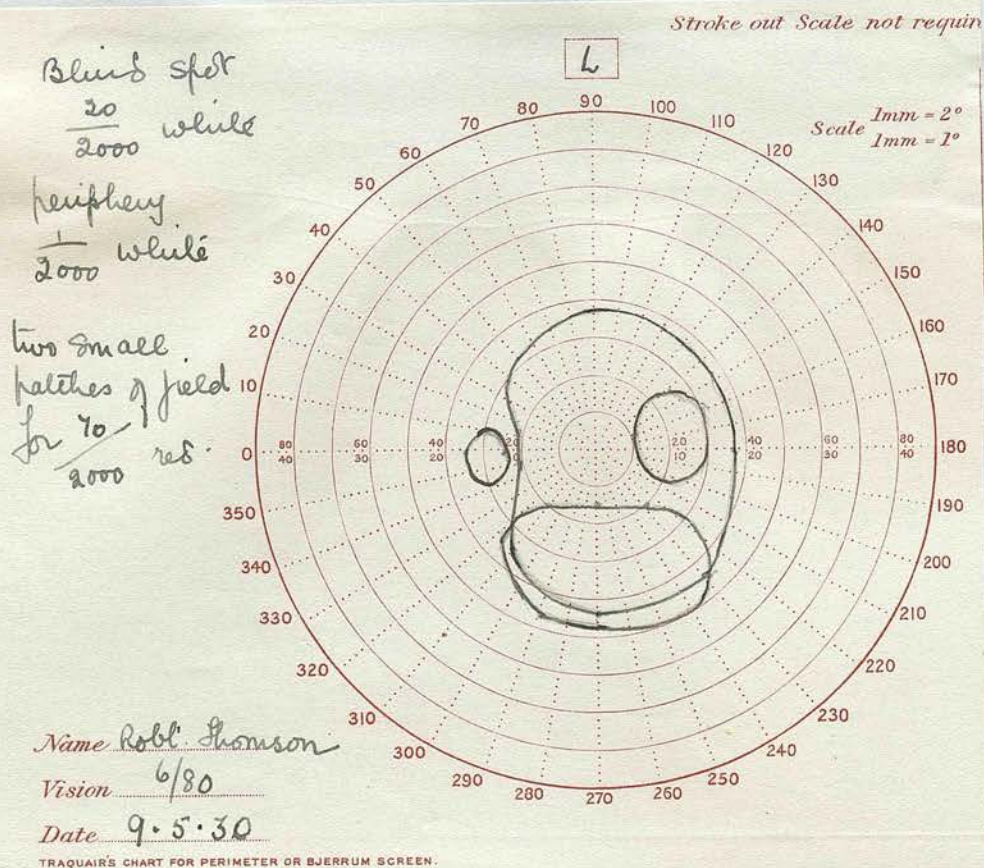
The commonest form that alcohol blindness takes is that of a very acute intoxication. It is caused by impurities. Shortly after taking the impure spirit the vision of both eyes begins to deteriorate and continues to do so very rapidly until vision is entirely lost. This is followed by recovery, and if the fields are charted at this time, the defect will be found to be pericentral and may include the blind spot. All stages of recovery are found, even to normal vision. Unfortunately a second stage often sets in and vision is again lost, this time permanently, and is accompanied by atrophic changes in the optic discs. One has no original case to illustrate this condition apart from alcohol cum tobacco.

ROBT THOMSON

d. Mark out blind spot.



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TRAQUAIRS CHART FOR PERIMETER OR BJERRUM SCREEN.

Case I.

Robert Thomson, 11 Blackwood Crescent. Age 55.

23.4.30. History: Eyes watering and sore.
Glasses not suiting. Smokes 4 to 5 ounces thick
black twist per week.

Examination: Wearing 3.5 D Sph. both eyes for
reading.

R.V. = $\frac{1}{2}$ c.r. no imp. L.V. = 6/80 no imp.
6/24

9.5.30. Pallor of temporal side of discs.
In both eyes fields for $\frac{1}{2000}$ white fell inside the
blind spot. For a red object nothing was present
save a small field in each eye for $\frac{70}{2000}$.

11.6.30.

R.V. = 6/12 L.V. = 6/24.

The peripheral field for $\frac{1}{2000}$ white object was
now full in each eye. There was a pericaecal
scotoma in each eye for $\frac{1}{2000}$ white. Normal blind
spot $\frac{20}{2000}$ white.

There was no red field present in either eye
for $\frac{40}{2000}$ red.

7.7.30.

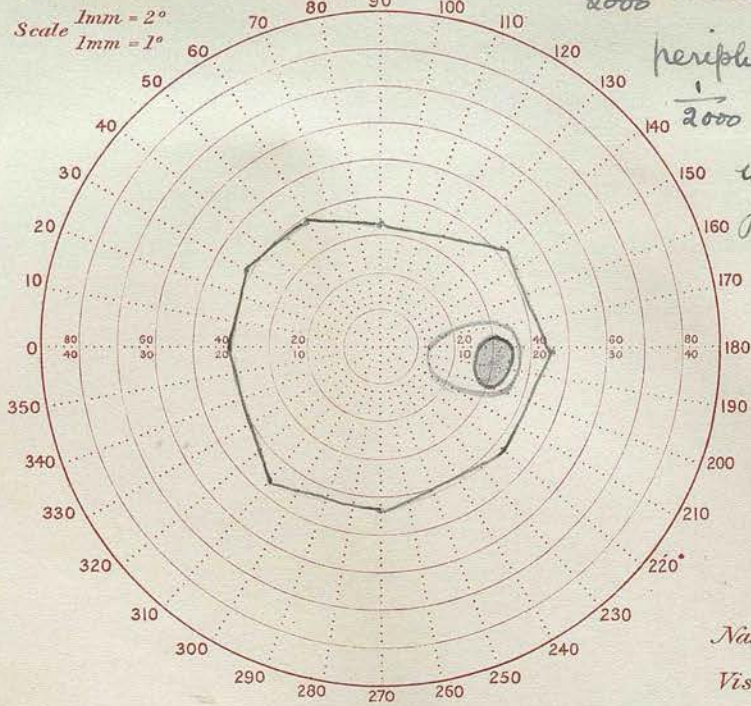
R.V. = 6/6 (-2) L.V. = 6/18 (-1)
 $\bar{c} + 1 = 6/12 (-1)$

Right field shows normal blind spot for $\frac{20}{2000}$

ROBT THOMSON

d. Mark out blind spot.

R

Blind spot
 $\frac{20}{2000}$ white.periphery scotoma
 $\frac{1}{2000}$ whiteno red field
for $\frac{40}{2000}$.Scale Imm = 2°
Imm = 1°

Name Robt. Thomson

Vision 6/24

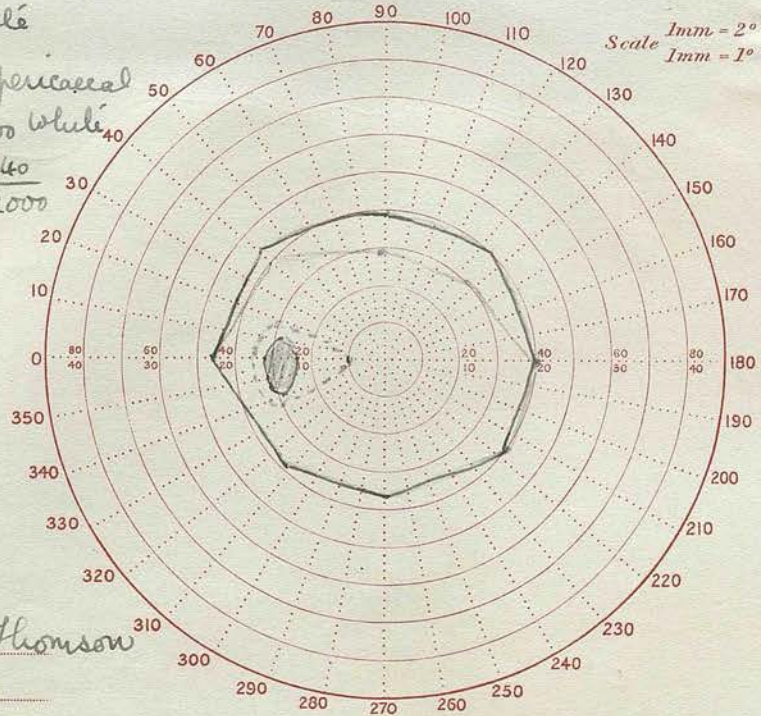
Date 11.6.30.

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Stroke out Scale not require

Blind spot
 $\frac{20}{2000}$ whiteperiphery + pericentral
scotoma $\frac{1}{2000}$ whiteno field for $\frac{40}{2000}$
red

L

Scale Imm = 2°
Imm = 1°

Name Robt. Thomson

Vision 6/12

Date 11.6.30.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

white and normal periphery for $\frac{1}{2000}$ white. Periphery for $\frac{5}{2000}$ red falls within the blind spot.

Left field shows normal blind spot for $\frac{20}{2000}$ white. Peripheral field falls within blind spot. Scotoma for $\frac{5}{2000}$ red.

4.9.30.

$$\text{R.V. } \bar{c} + 0.5 = 6/6$$

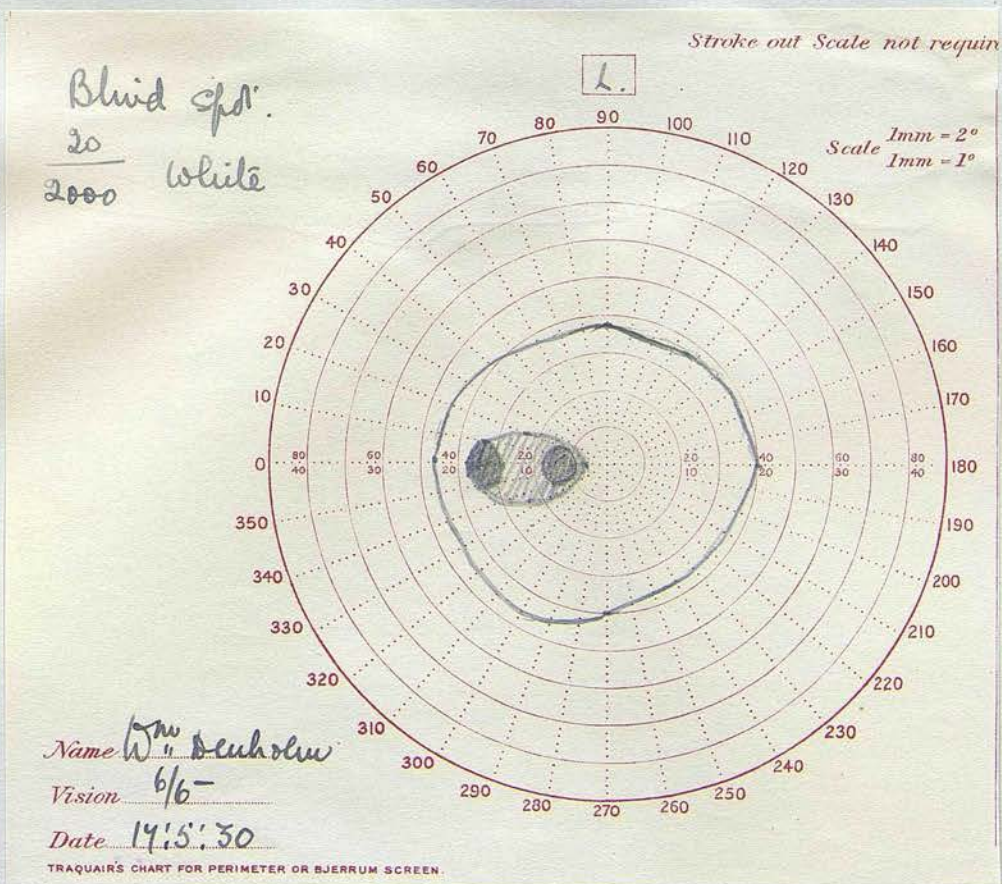
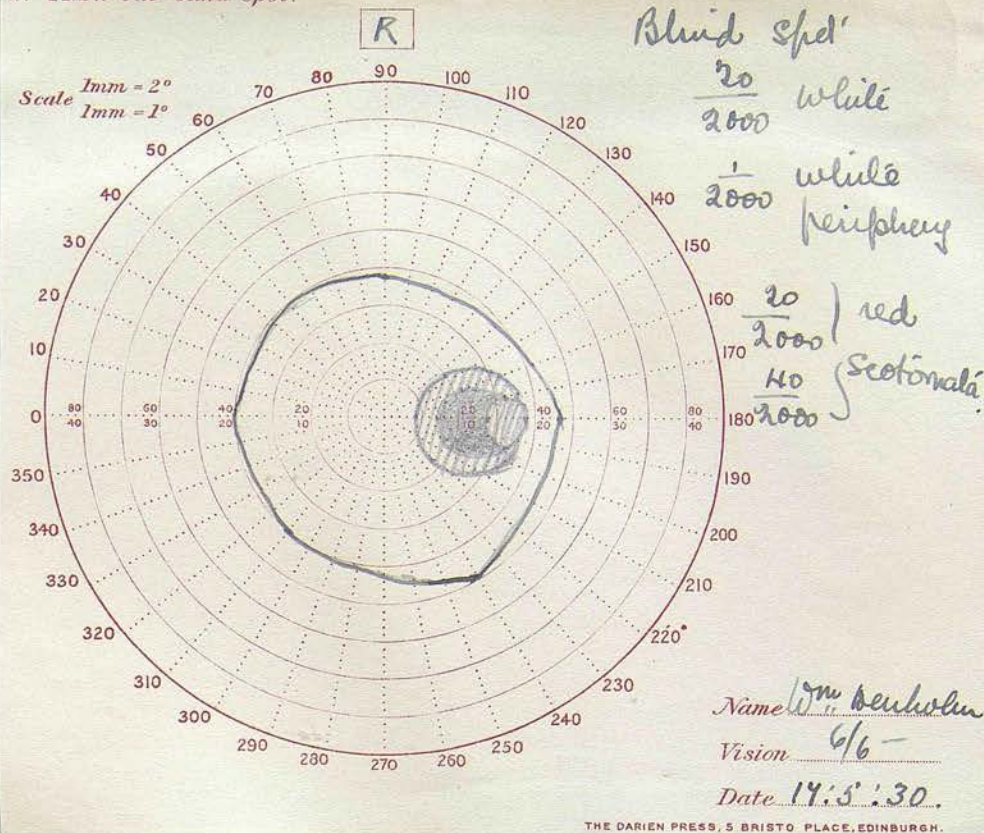
$$\bar{c} + 3 = J 1$$

$$\text{L.V. } \bar{c} + 0.5 = 6/9 (-1)_{180^\circ}$$

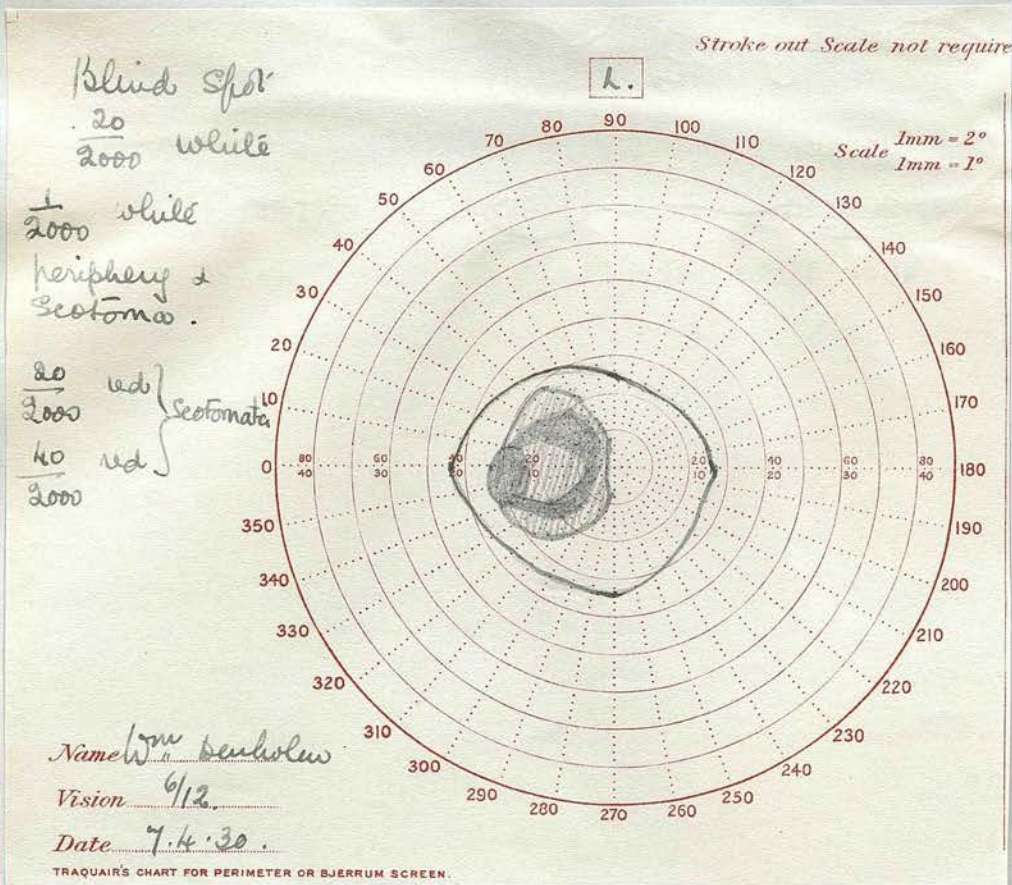
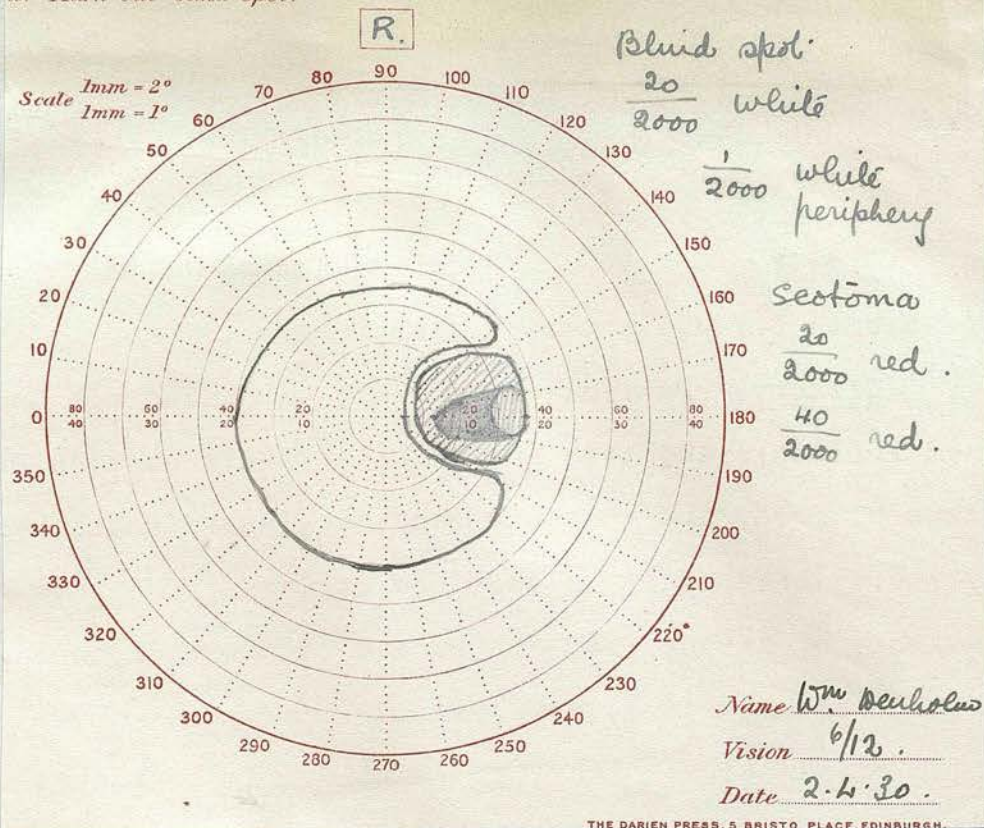
$$\bar{c} \begin{array}{l} + 2.5 \\ + 0.5 \end{array} = J 2_{180^\circ}$$

No field defects.

d. Mark out blind spot.



d. Mark out blind spot.



Case II.

William Denholm, 152 Bonnington Road. Age 59.

1.4.30. Complaint: Blurred vision in both eyes both for distance and near.

History: Patient stated that six weeks ago he had influenza and that after that objects outside became blurred, and that his reading glasses which had formerly been suitable were now useless. He gave a history of having smoked 4 ounces per week of strong mixed tobacco for several years.

Examination: Wearing + 2.25 D Sph. both eyes for reading. Very little refractive error. Fundi normal.

R.V. = 6/12	L.V. = 6/12
C + 2.5 J 12	B.E.

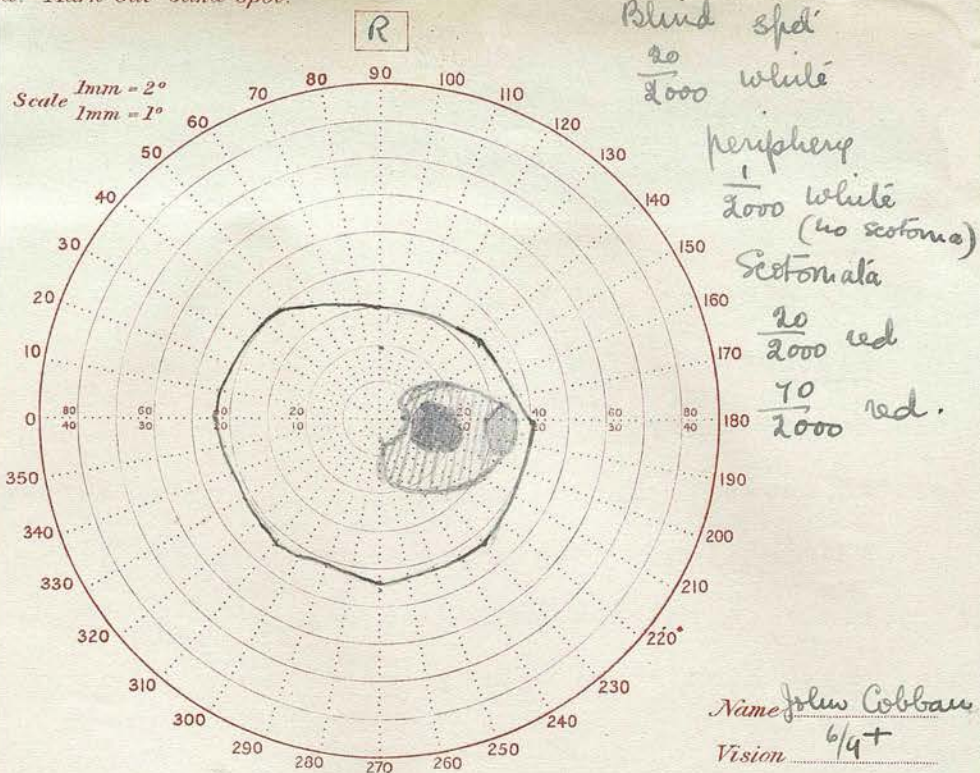
No improvement with glasses.

2.4.30. Examination of field of vision of right eye showed that the field for 1/2000 white fell inside the blind spot and came close up to the fixation point. A centrocaecal scotoma for 20/2000 red encircled the blind spot and came close up to white field, and 40/2000 red showed a small scotomatous tongue stretching from blind spot towards the fixation point.

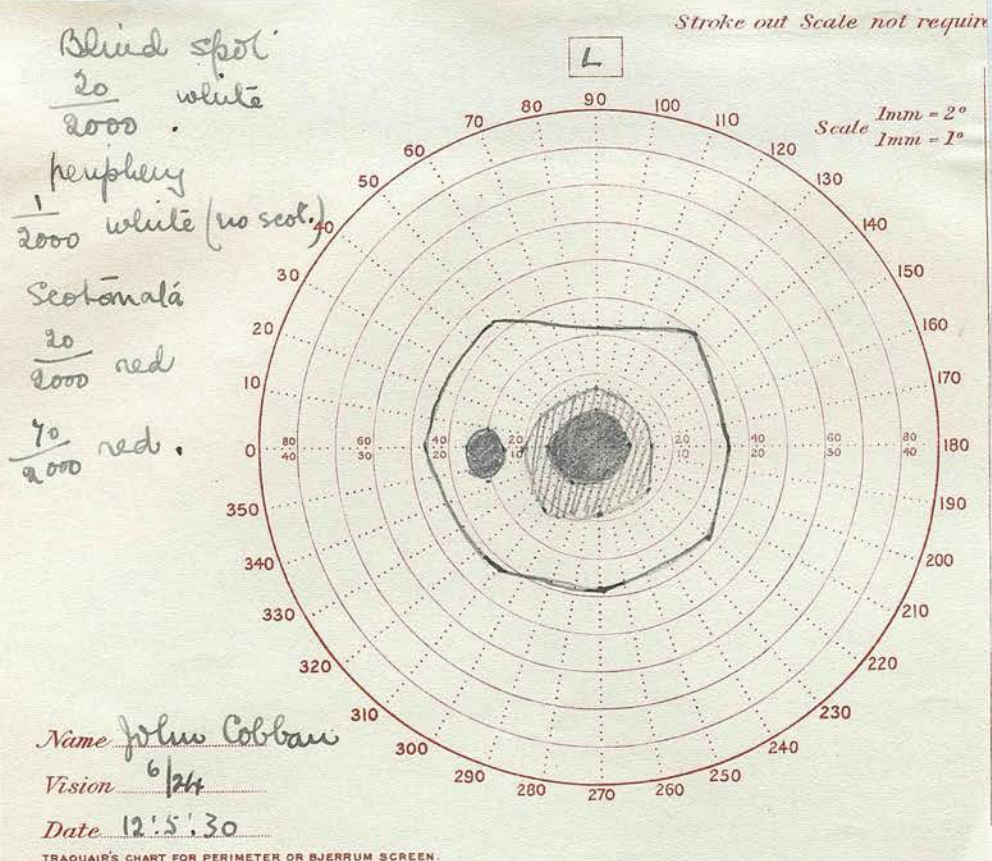
17.5.30. The field for 1/2000 white now showed as full in each eye, and although scotomata for 40/2000 and 20/2000 red were still present, they had diminished considerably in size.

R.V. = 6/6 -	L.V. = 6/6 -
--------------	--------------

d. Mark out blind spot.



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Case III.

John Cobban, 40 Hawthornvale. Age 40.

22.4.30. Complaint: Hazy vision for two weeks both for distance and reading.

History: Patient states that he has been unable to see clearly for either distance or reading for two weeks. Smokes 3 ounces Irish roll per week.

Examination: Discs have a peculiar neuritic appearance, with very congested veins. This, however, was taken to be physiological.

12.5.30. R.V. = 6/24. L.V. - 6/9 +

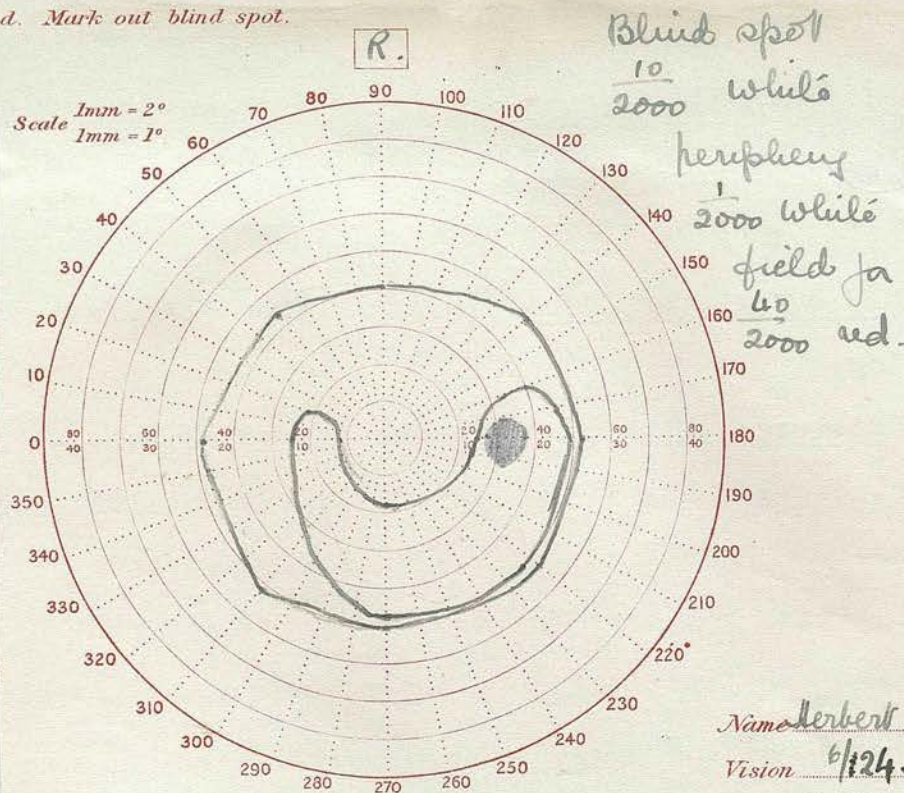
Both fields showed full periphery for 1/2000 white. In each eye there were scotomata for 20/2000 red - 70/2000 red. In the right eye these did not overlap the fixation area, which accounted for the better vision.

In view of the congestion state of the retinal veins and cyanotic hands, patient was examined by a physician who reported as follows:-

B.P. 140/100 Apex beat 6th interspace $\frac{1}{2}$ " interval to nipple line. All sounds closed. No irregularity. Response to effort good.

Field of vision for 1/2000 white was full. In both eyes scotomata for 20/2000 red and 70/2000 red was present. In the left eye they overlapped the fixation area and the vision was poor, while in the right eye the fixation area was spared and the vision was much better.

ed. Mark out blind spot.



Name Herbert Cobb.

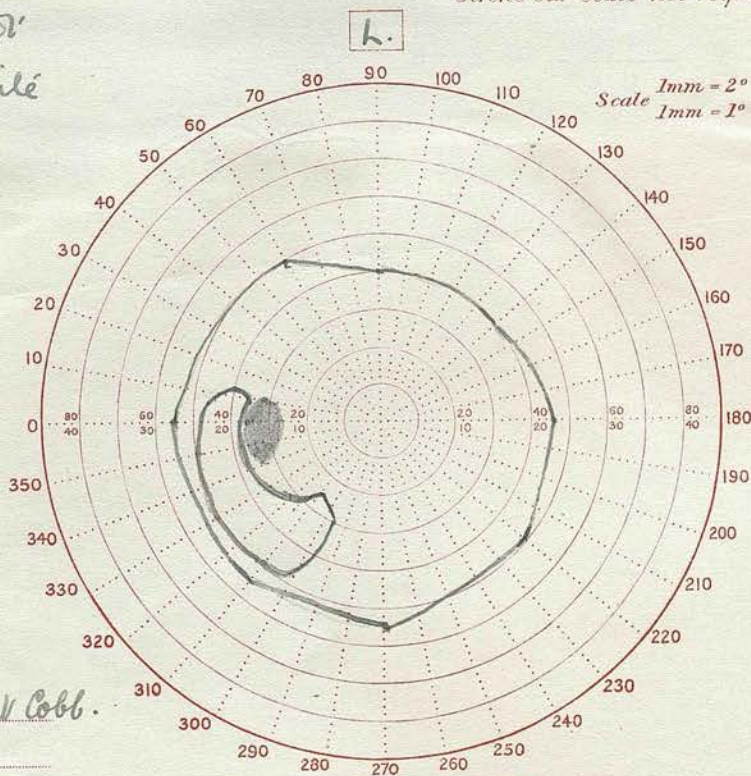
Vision 6/24.

Date 19:3:30.

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Stroke out Scale not required

Blind spot
 $\frac{20}{2000}$ white
periphery
 $\frac{1}{2000}$ white
field ja
 $\frac{40}{2000}$ red.



Name Herbert Cobb.

Vision 6/36

Date 19:3:30.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Case IV. Tobacco Amblyopia from Cigarette Smoking
cum Alcohol.

Herbert Cobb, 17 Dudley Crescent, Leith. Age 41.
Lodging House Keeper.

18.3.30. Complaint: Misty vision for distance
reading.

History: Four to six weeks ago patient began
to notice that his vision was misty for both distance
and near. He stated he never had any previous
trouble with his eyesight and he has never had
glasses. He is probably a chain smoker of cigarettes
70 - 80 per day, Woodbine and Craven A.

Examination: Pupils react sluggishly to light,
but well to convergence.

R.V.	L.V.
$\bar{c} \frac{+1}{+0.5} = 6/36 \quad C \ 6$ <p style="text-align: center;">90°</p>	$\frac{+1}{+0.5} = 6/24 \quad C \ 6$ <p style="text-align: center;">90°</p>

Fundi appear normal.

Temporal part of disc is pale, but not unduly so.

Field changes are those of toxic amblyopia.

2.4.30. R.V. c gl. - 6/18 slowly.

L.V. c gl. - 6/24 slowly.

Fields appear practically the same.

19.9.30. R.V. $\bar{c} + 1.5 = 6/9$ L.V. = 6/9.

B. RETROBULBAR NEURITIS.

Retrobulbar neuritis is an inflammatory condition of the optic nerve and for purposes of this thesis may be divided into two groups:-

- (1) Axial (acute and chronic)
- (2) Total transverse.

Of these the first is important from the point of view of central failure of the field of vision, and the second may be looked on as a very severe variety of the first.

Retrobulbar neuritis also occurs, but more rarely, in the chiasma, and still less frequently in the tract.

(1) ACUTE AXIAL NEURITIS.

This is the commonest form of retrobulbar neuritis seen by the ophthalmic surgeon. It chiefly occurs in youth and early middle age and is characterised by a rapid unilateral failure of vision often associated with pain in the region round the eye, with pain on movement of the eye and tenderness on pressure. The onset is rapid and the recovery slow. On examination the pupil contracts

directly to light, but the contraction is not held. The consensual reaction is, however, normal. The fundus is usually normal, but there may be slight evidence of optic neuritis. The vision may be only slightly depressed, but it is more common to find considerable depression of vision especially central.

Field Changes.

The typical field change is a loss of central vision. This is most commonly pericentral, but it may also be paracentral, or centro-caecal.

The shape is usually round, but may be oval or irregular, and the size varies.

The scotoma may be absolute or, on the other hand, very slight and considerable variations of intensity are found.

Sometimes denser nuclei are found within the scotoma. The edges are usually steep. The onset is sudden and the recovery gradual. Disproportion between colour loss and white loss indicates a rapidly progressing condition, whether during the attack or during the recovery.

Pathology.

Inflammation may take place in the optic nerve in any part of its course. When the inflammation involves the nerve below the entrance of the retinal

vessels, the familiar appearance of optic neuritis in the disc is visible with the ophthalmoscope. When the inflammation lies behind this area, it is called retrobulbar neuritis and no change is seen in the disc with the ophthalmoscope at the time, although a descending atrophy may appear later.

Clinical Significance.

In a case of retrobulbar neuritis the failure of vision is frequently the only symptom complained of and the field changes the only signs found. This being so, it behoves us to make a very careful examination of the visual fields of both eyes in order to throw light on the two points which require elucidation, namely, the position and cause of the lesion.

Position: It is most common to find the field changes confined to one eye, and these indicate the presence of a lesion in one optic nerve below the chiasma. The appearance of the disc head must be carefully noted. Commonly the disc appears normal, but in some cases there is obscuration of the edges and swelling. In the case of a very small central scotoma, it is necessary to exclude a macular lesion.

If hemianopic characters are found in the central scotoma, this means that the lesion is situated at a point where the crossed and uncrossed macular fibres are separated, i.e. at the chiasmal end of

the nerve or beginning of tract. These have been called "junction" Scotoma (Traquair).

A lesion situated in the chiasma involves both fields and gives rise to bilateral hemianopic defects. These are not necessarily at the same stage.

Cause. If the case follows the typical description regarding age of patient, rapid onset, with pain and profound depression of vision, there is little doubt but that we are dealing with an inflammatory condition. Very many of the cases later develop disseminated sclerosis. In fact, disseminated sclerosis has been cited as the cause of retrobulbar neuritis in as high a figure as 40% of cases. See reference from Lancet under heading 'Disseminated Sclerosis'.

Disease of the accessory sinus, whether active or "latent", has been thought by some to account for a high percentage of cases. The active group may account for a few rare cases, the "latent" for probably none. This will be referred to again under the heading of accessory sinuses.

In other cases septic teeth have been found, and as the condition cleared up after the removal of the teeth, these were put down as the cause. As a matter of fact these cases almost invariably get better without any treatment whatsoever. It is necessary, however, to leave no stone unturned

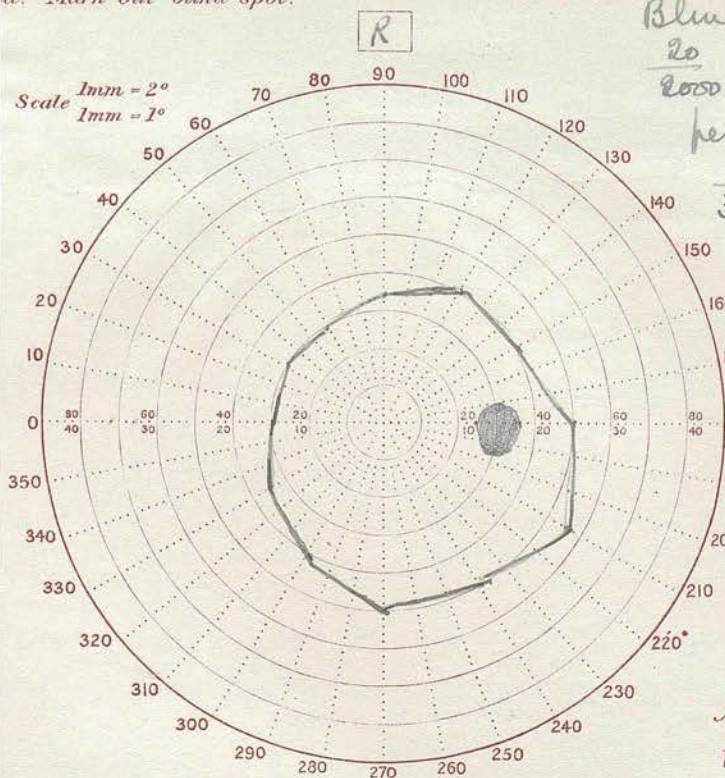
to find a cause and to place the patient in a satisfactory condition of general health. In some cases the neuritis is bilateral and usually shows changes in the disc head with the ophthalmoscope. The cause in these cases is just as difficult to find, but may be presumed to be of systemic origin - Leber's disease, myelitis or influenza may be responsible.

Prognosis in the common rapid unilateral variety is good as regards sight.

If the onset has been slow, and especially in the bilateral cases, considerable depreciation of vision results.

MRS COWE

d. Mark out blind spot.



Blind spot
 $\frac{20}{2000}$ white
periphery
 $\frac{1}{330}$ white
no Central
Scotoma for
 $\frac{1}{2000}$ white,
red or green.

Name Mrs Cowe

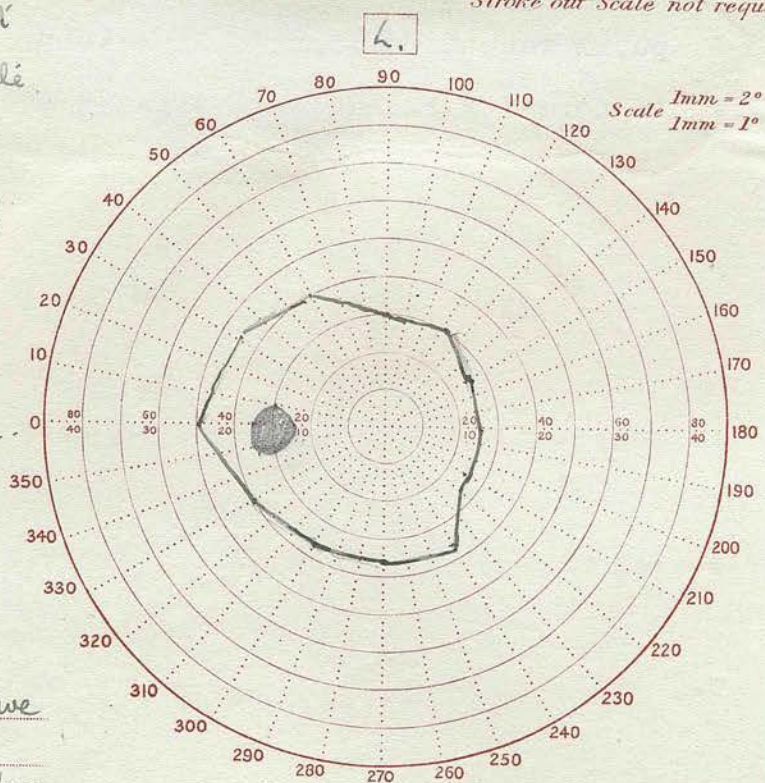
Vision 6/6

Date 14/X/29

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Blind spot
 $\frac{20}{2000}$ white
periphery
 $\frac{1}{330}$ white

no Central
Scotoma for
 $\frac{1}{2000}$ white,
red or green.



Name Mrs Cowe

Vision 6/6

Date 14/X/29

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Example of Typical Case, with Complete
Recovery. Cause unknown.

Case I.

Mrs Cowe, aged 35.

11.6.24. Complaint: Failure of vision left eye.

History: Patient first noticed that the sight of the left eye was failing ten days previously. The failure came on suddenly and progressed rapidly, and was accompanied by pain in the region of the eye and headache. No history of any recent illnesses of importance.

Examination: R. pupil, normal reaction.
L. pupil reacts to light, but reaction not held.
Fundi normal examined with dilated pupils.

R.V. $\bar{c} + 1 = 6/6$. L.V. = F. close.

R. field no central scotoma.

L. field central scotoma present.

13.6.24. Examined by rhinologist, who reported only abnormality was deviated septum. This was resected on 26/6/24. X-ray negative. Wassermann reaction negative. Vision began to improve.

17.7.24. R.V. = $6/6 \bar{c} + 1$. L.V. = 6/18.

10.6.25. R.C. $\bar{c} + 1 = 6/6$. L.V. $\bar{c} + 1 = 6/6$.

14.10.29. Seen five years later. Age 40.

$$R.V. = 6/12 \bar{c} + 1.25 = 6/6$$

$$L.V. = 6/9 + \bar{c} + 1 = 6/6.$$

Add + 0.5 J 1 B.E.

Pupillary reactions normal. Right pupil if anything a little larger than left. Fundi normal. No sign of optic atrophy. Peripheral fields of both eyes normal for 1/330 and 1/2000 white. No central scotoma either eye for 1/2000 white, red or green.

No other signs of disseminated sclerosis. Patient has been well during the last five years. Is to all intents and purposes in perfect health.

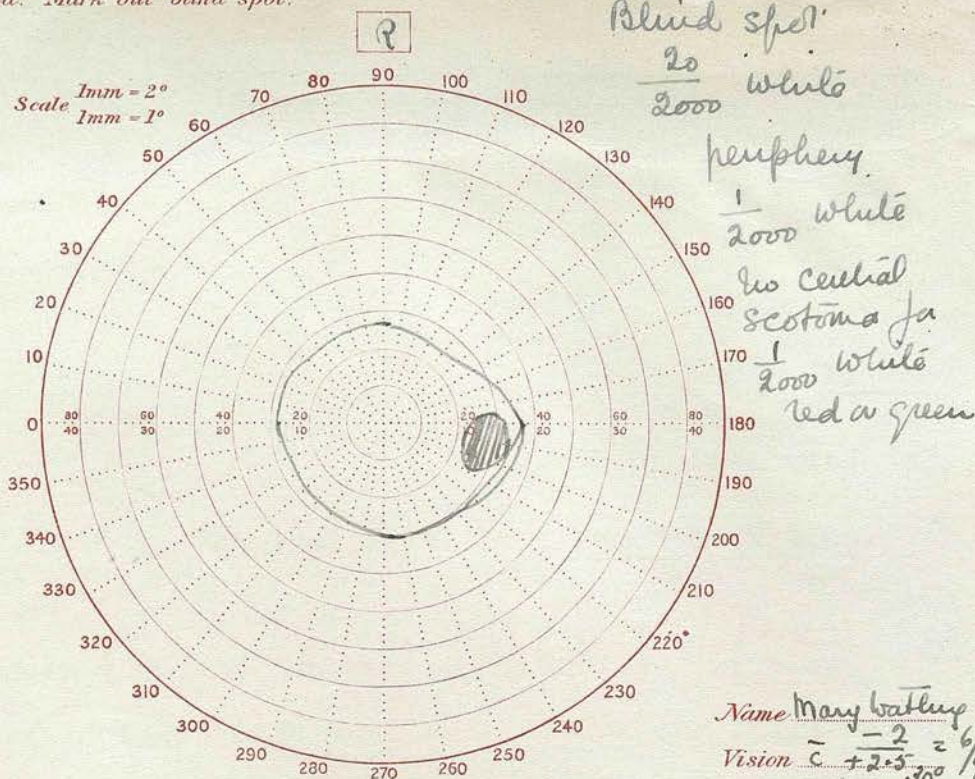
Summary.

This is an example of the group of cases which baffles all attempt to find a cause. From the original examination of fields of vision, it was found that there was involvement of the left optic nerve below the chiasma. The condition was obviously a very acute inflammation as seen from the rapidity of onset and profundity of loss. Thorough examination disclosed no abnormality except a deviated septum, which was resected. Although vision commenced to improve immediately after the operation, it is impossible to think that a condition which

must have been there for years can have been the cause of so sudden and acute an inflammation. Five years later patient was again thoroughly examined. No abnormality was found in the fields of vision, there were no signs of disseminated sclerosis, and patient was at time of examination and had been for the previous five years in perfect health.

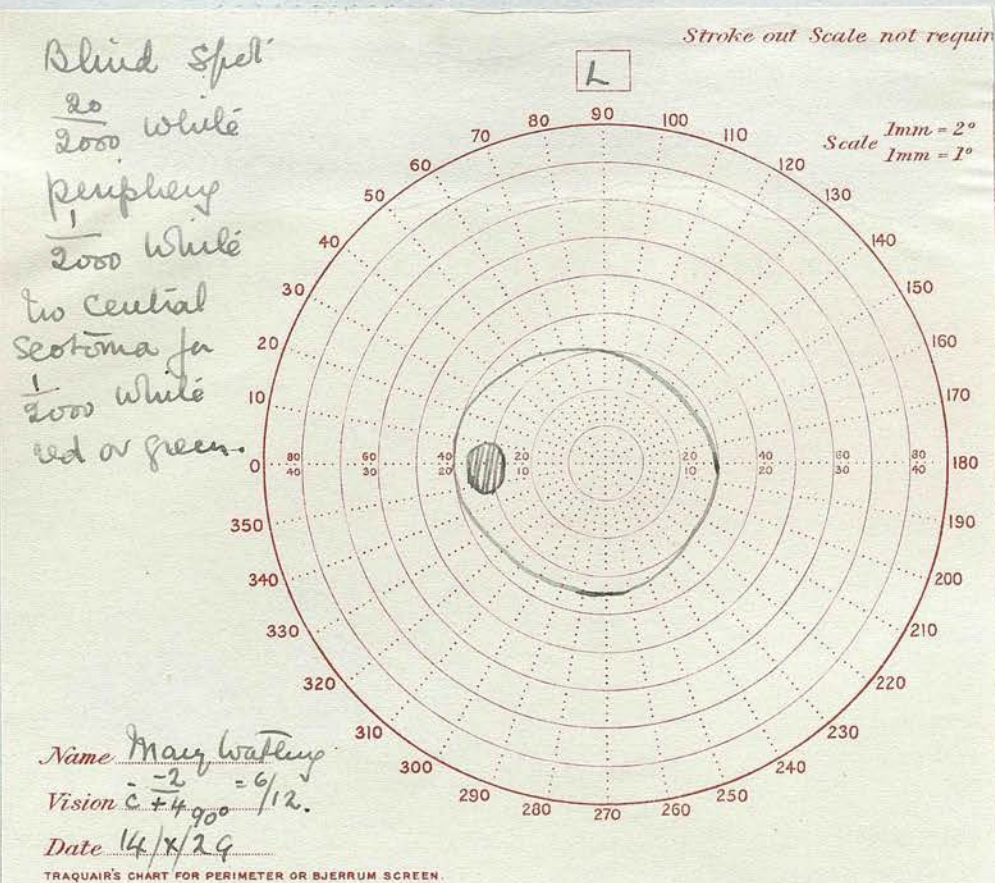


ed. Mark out blind spot.



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MARY WATLING



TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Example of Typical Case,
with Complete Recovery.

Case II.

Mary Watling. Age 19.

30.6.25. Complaint: Mist in front of right eye.

History: Patient first noticed that the sight of the right eye was failing four days previously. The failure came on suddenly and progressed rapidly. There were no previous illnesses.

Examination: Right pupil reacts to light, but reaction not held.

Left pupil, normal reactions.

The fundi were healthy when examined with dilated pupils. Opaque nerve fibres present in right eye.

R.V. = F at $\frac{1}{2}$ m.

L.V. with glasses = 6/18 + (refractive error present).

Right field showed central scotoma.

Left field normal.

1.7.25. Examined by rhinologist who reported

"No tenderness over floor of frontal sinuses. On Anterior Rhinoscopy there is no great anterior nasal obstruction; septum to right high up.

"Tonsils not specially enlarged; anterior pillars red.

"Definite adenoid mass.

"Tympanic membranes are normal.

"There is no sinus disease so far as can be seen from nasal examination and transillumination."

X-ray report negative.

No operation was performed.

3.9.25. R.V. = 6/80 L.V. with glasses = 6/18+

7.7.25. R.V. = 6/36 L.V. ditto.

10.7.25. R.V. = 6/18 L.V. ditto.

14.7.25. R.V. = 6/12 L.V. ditto.

Seen five years later. Age 25.

$$\text{R.V. } \bar{c} + \frac{-2}{2.5} = 6/9 \\ 80^{\circ}$$

$$\text{L.V. } \bar{c} + \frac{-2}{4} = 6/12. \\ 90^{\circ}$$

Patient has been perfectly well since this attack of retrobulbar neuritis.

Right field shows no central scotoma or other abnormality. There are no signs of disseminated sclerosis.

Summary.

In this case the patient had absolutely no treatment and recovered her sight perfectly. The records of vision as 6/9 and 6/12 are to be explained by the high degree of mixed astigmatism.

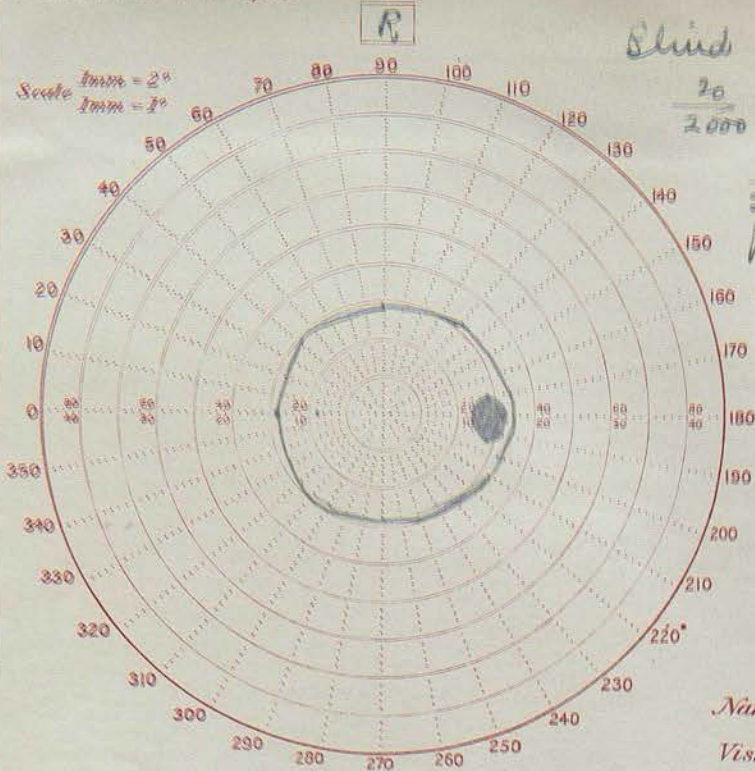
Although there are no signs as yet of disseminated sclerosis, this is a case which might still turn out to be that disease, as the patient is as yet only 25.

$$\text{R.V. } \bar{c} \text{ gls.} = 6/6. \quad \text{L.V. } \bar{c} \frac{-0.5}{+2} = 6/12.$$

CASE IV ACUTE RETROBULBAR NEURITIS
JOHN LIVINGSTONE

opp.

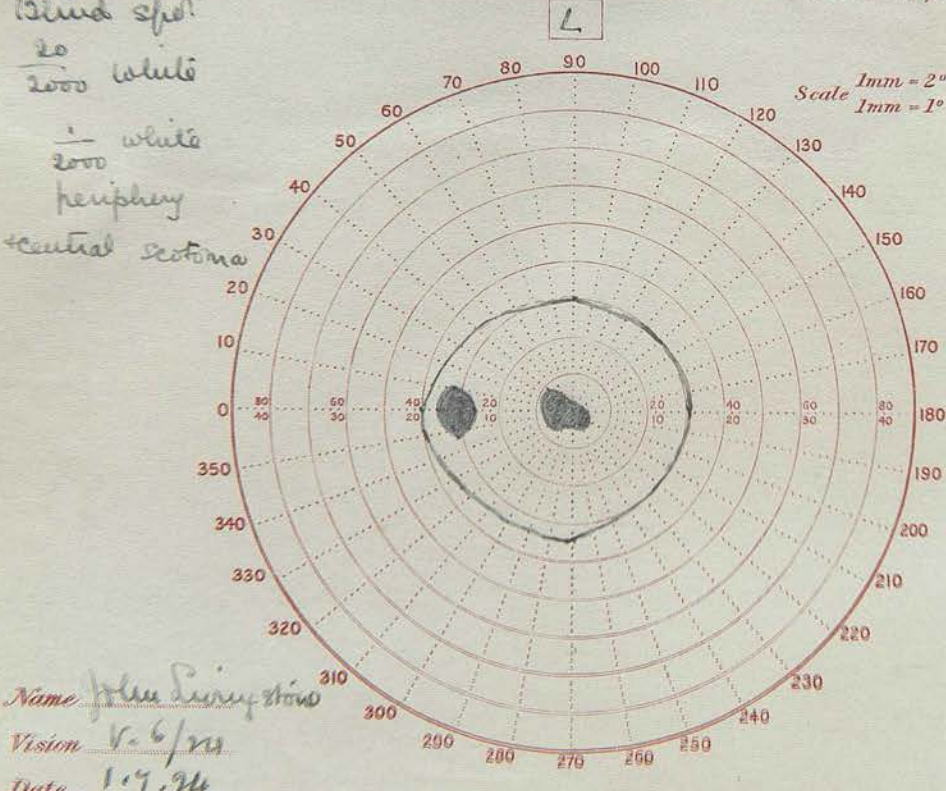
ed. Mark out blind spot



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Blind spot
20
2000 white
2000 white
periphery
central scotoma

Stroke out Scale not required



TECHNIQUE'S CHART FOR PERIMETER OR BJERRUM SCREEN

Case IV.

John Livingstone, 6 Leslie Place, Shotstown,
Penicuik. Age 28.

History. 15.6.29. History of blow from piece of stone on left eye. No evidence of injury. Pain at side of eye.

Examination: Pupil reacting well. Central failure.

R.V. = 6/6. L.V. = 6/60.

18.6.29. R.V. = 6/6 & J 1. Left pupil reacts to light, but reaction not held. Oedema of left optic disc.

27.6.29. R.V. = 6/6. Pupil reacts well. Still oedema. L.V. = 3/60.

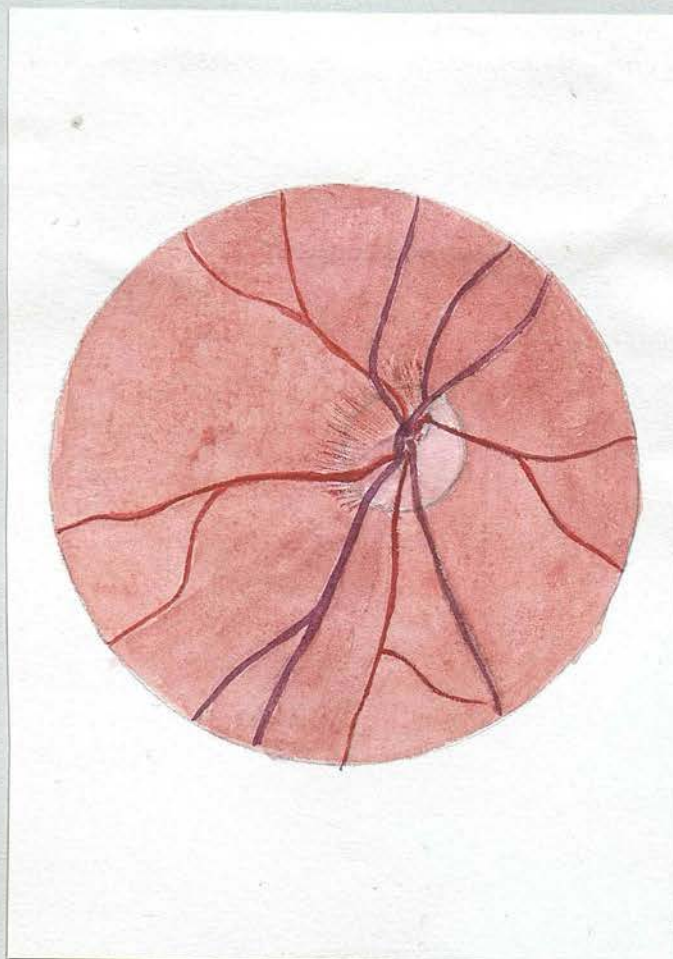
1.7.29. R.V. = 6/6. L.V. = 6/24. Field taken. Central scotoma present.

13.7.29. Disc pale. Vision unchanged 6/24. W.R. negative.

20.7.29. R.V. = 6/6. L.V. = 3/60. Optic atrophy.

Summary.

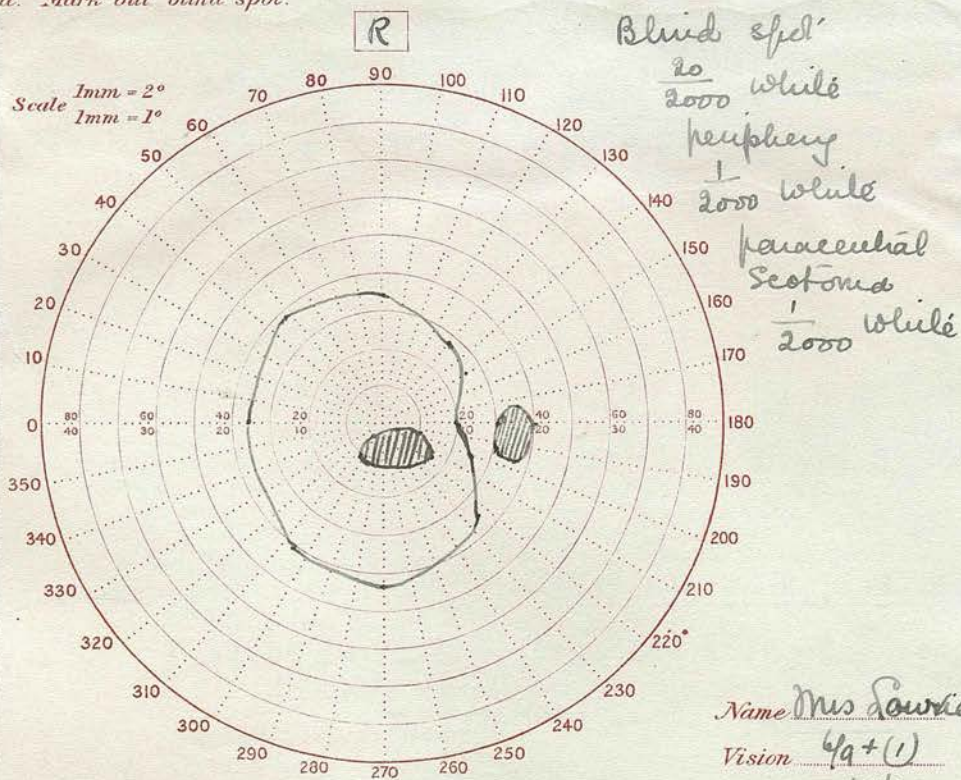
Cases numbers III. and IV. have not been seen again. They are both typical cases of retrobulbar neuritis from no obvious cause occurring in young people. Case III. had improved as regards vision on the occasion of the last visit, but Case IV.

CASE OF ACUTE UNILATERAL RETROBULBAR NEURITISJOHN LIVINGSTONE

illustrates the type of case in which the vision does not improve and the end result is optic atrophy and very poor vision.

MRS LOWRIE

d. Mark out blind spot.



Case V.

Mrs Elizabeth Lowrie, 8 George Street, Eyemouth.
Age 24.

15.5.30. Complaint: Misty vision in right eye.

History: Patient complains that one week ago she suffered from a pain in the right eye and mistiness of vision, with headache.

Examination: Patient has pain on moving the right eye and also on palpation.

R.V. = 6/28. L.V. = 6/9 $\bar{c} + 0.75 = 6/6$.

Right disc shows blurring of disc margins and congestion of veins.

Ear, Nose and Throat Report: A little pus in nose. Tonsils enlarged. Removal advised. X-ray.

16.5.30. Small haemorrhage.

R.V. = 6/9.

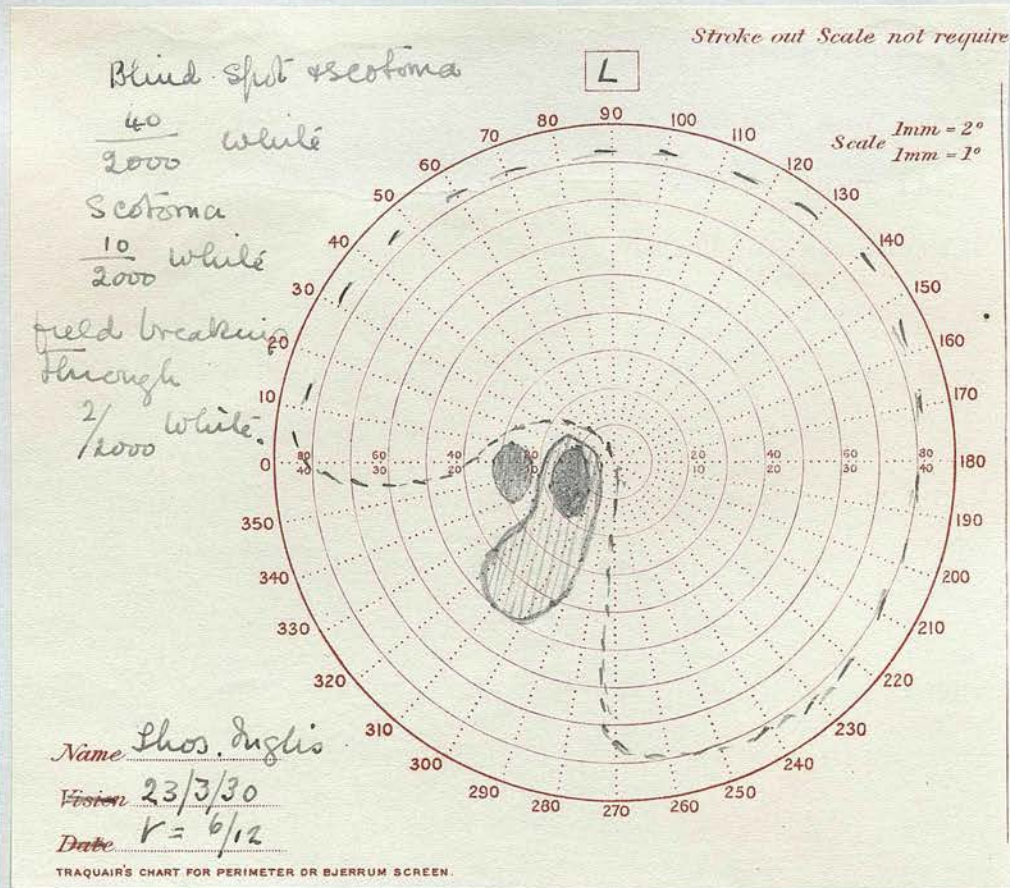
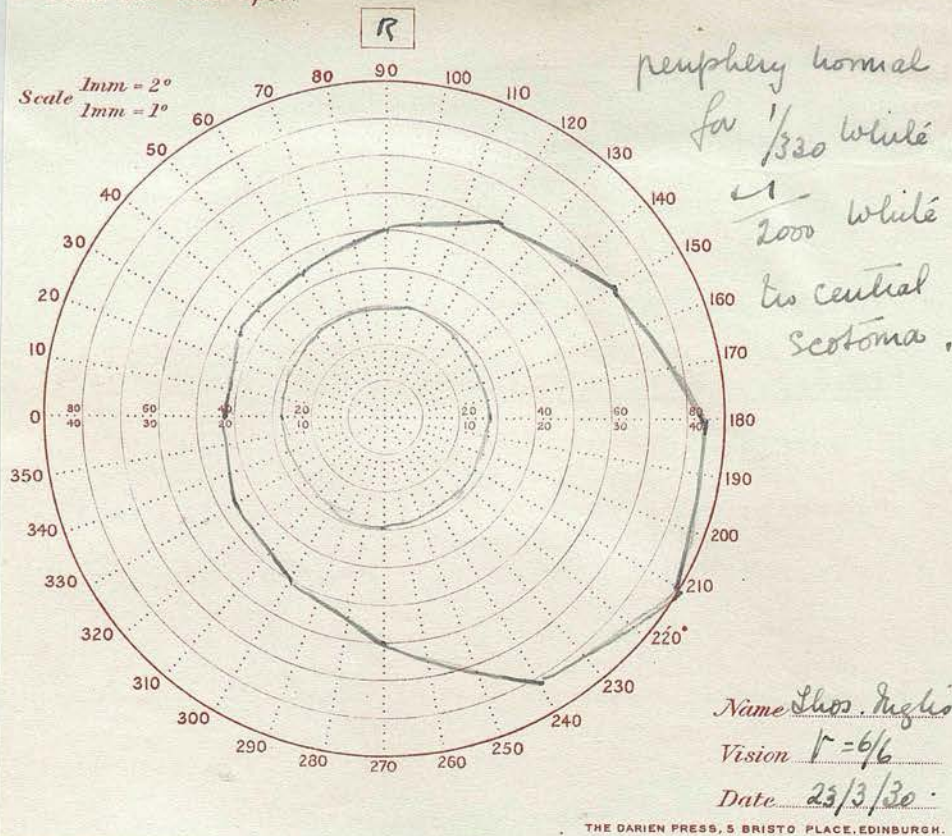
Fields of vision showed a paracentral scotoma for 1/2000 white.

17.5.30. V. = 6/9- J 1 L.V. = 6/9+ J 1.

Summary.

Typical case showing almost perfect recovery.

d. Mark out blind spot.



Case of Recurrent Retrobulbar Neuritis.Case VI.

Thomas Inglis, 20 Hawthorn Vale. Age 36.

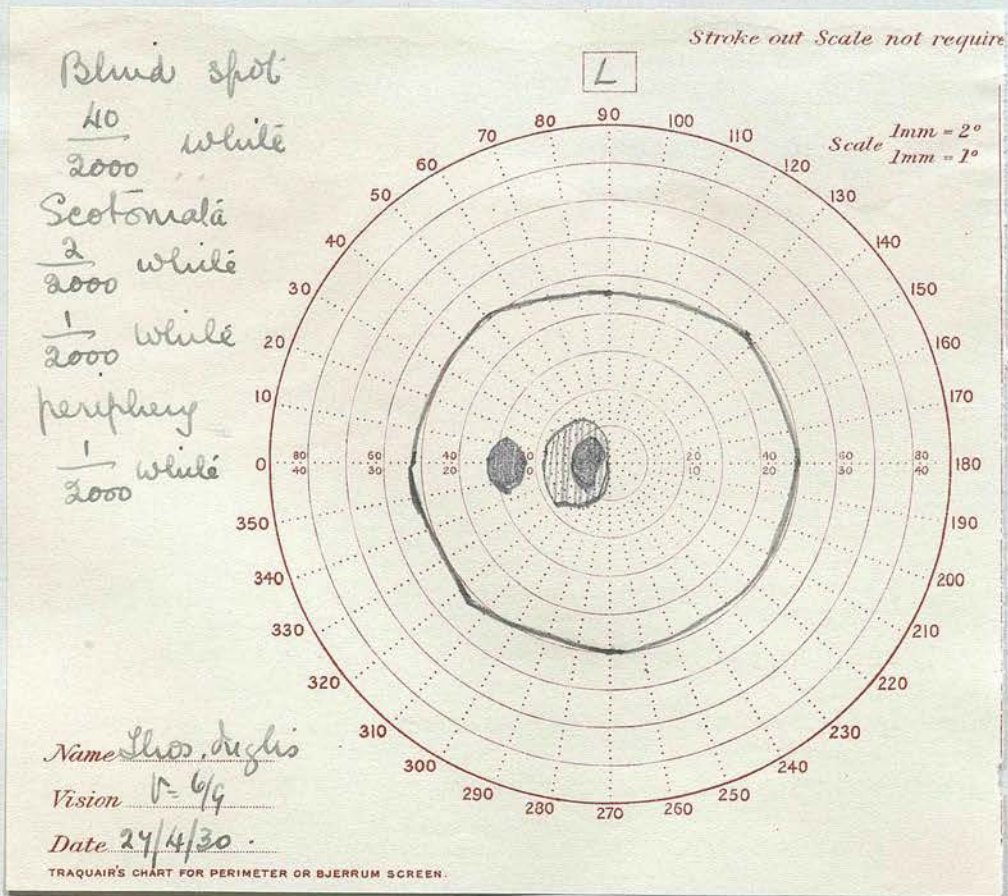
18.3.30. Complaint: Failure of vision left eye. Duration one week.

History: One week ago vision of left eye failed fairly rapidly. There has been no improvement. The failure of vision was unaccompanied by any pain, but patient has recently had a bad cold. Patient stated that in October, 1921, he lost the sight of the left eye very rapidly. The loss in this case was profound, patient being only able to distinguish light from dark. Patient does not remember if there was any pain. The vision returned gradually until perfect vision was attained, according to his statements. Unfortunately the eye record of this case cannot be found, but evidently a thorough examination was carried out and the notes of the rhinological examination are available.

Ear, Nose and Throat Notes:

Ant. Rhin. M.M. very congested. Swelling of left inferior turbinal: no pus. Large spine on right of septum. Both middle and interior right turbinals congested.

Post. Rhin. Postnasal discharge, with caking of mucus on posterior wall. Choanae clear.



20.10.21. X-ray indefinite. Mucopus in sphenoidal recess.

31.10.21. Nasal submucous resection.

3.11.21. Satisfactory.

Ophthalmological Examination:

18.3.30. Vision of right eye has never been quite so good as that of left, but with R.V. $\bar{c} + 2 = 6/6$ J 1. Right pupil reacts to light and holds reaction very well. Right fundus normal.

Left vision = 6/12. No improvement with glasses. Left pupil reacts, but does not hold reaction well. Left disc is distinctly pale.

19.3.30.) Fields taken show no sign of affec-
23.3.30.) tion of right eye either for 1/330
 or 1/2000 white.

Left eye shows depression of whole field for 1/330 white, and paracentral scotoma in lower temporal quadrant for 2/2000, 10/2000 and 40/2000 white.

20.3.30. Wassermann reaction negative.

20.3.30.) X-ray. A.P. and lateral show no
26.3.30.) abnormality.

Knee-jerks brisk.

No diplopia, nystagmus, or intention tremor.

27.4.30. R.V. $+ 2 = 6/6$. L.V. $\bar{c} + 2 = 6/9$.
 Field again charted.

Peripheral field for 1/2000 now normal.

Scotomata present for 1/2000 white and 2/2000

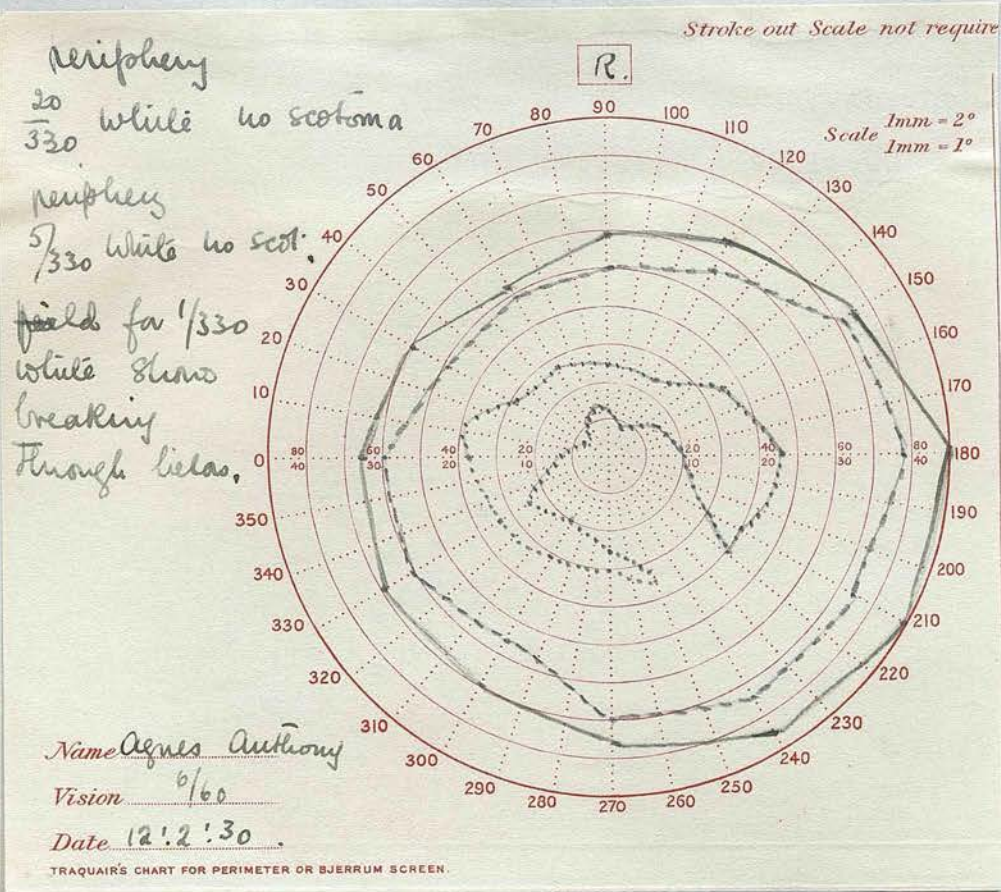
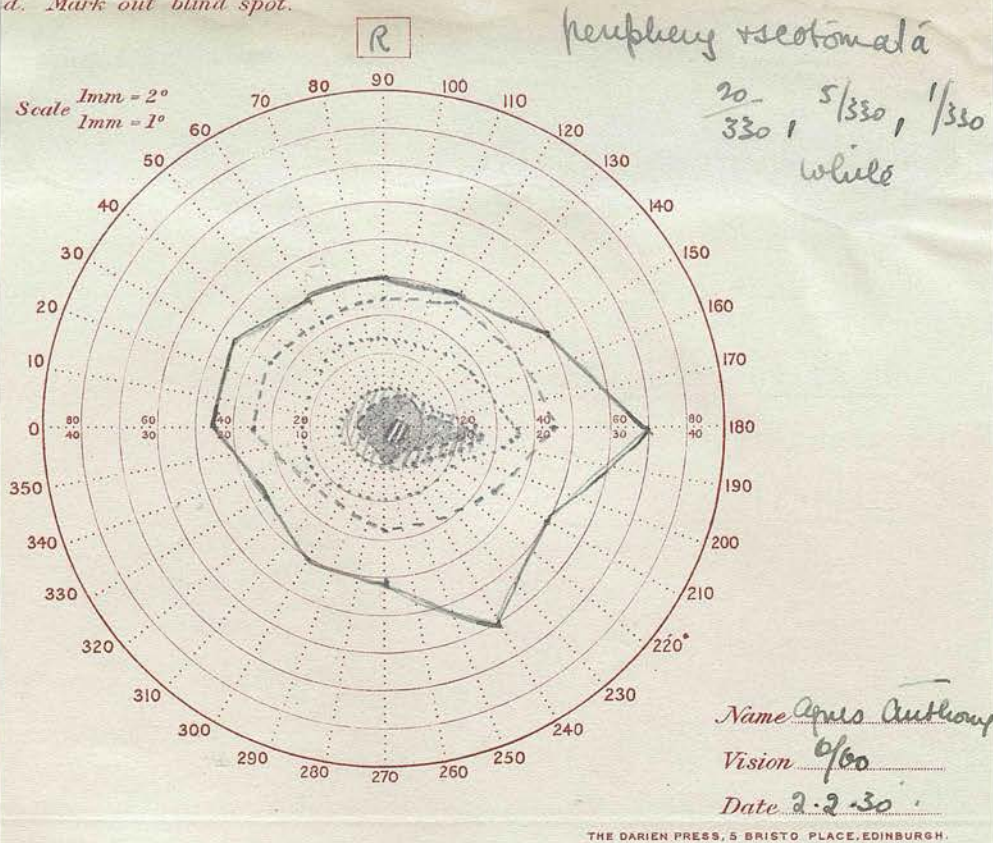
white lying between fixation point and blind spot;
that for 1/2000 comes close up to fixation point.

Note R.V. = 6/9.

Summary.

This case is distinctly uncommon in that the patient had two attacks of retrobulbar neuritis in the same eye, and in spite of that and some pallor of nerve head, vision recovered to 6/9.

d. Mark out blind spot.



Case VII.

Agnes Anthony, Farm Worker, South Pitdinnie,
Cairnhill. Age 20.

25.11.29. Complaint: Pain in left eyeball for
three weeks, especially on movement.

Examination:

0.25 c.r. 90°	0.25 c.r. 90°
V. = 6/6 & J 1.	V. - 5/60

Discovered defect five days ago.

Pain on palpating. Tenderness.

Oedema of upper optic disc.

Retrobulbar neuritis.

Scotoma of centro-caecal area and lower outer
quadrant.

29.11.29. Admitted to Ward.

1.12.29. V. - 6/6 J 1. More swelling of disc.
Oedema round inner half and a little below. V. =
C F at 8 in.

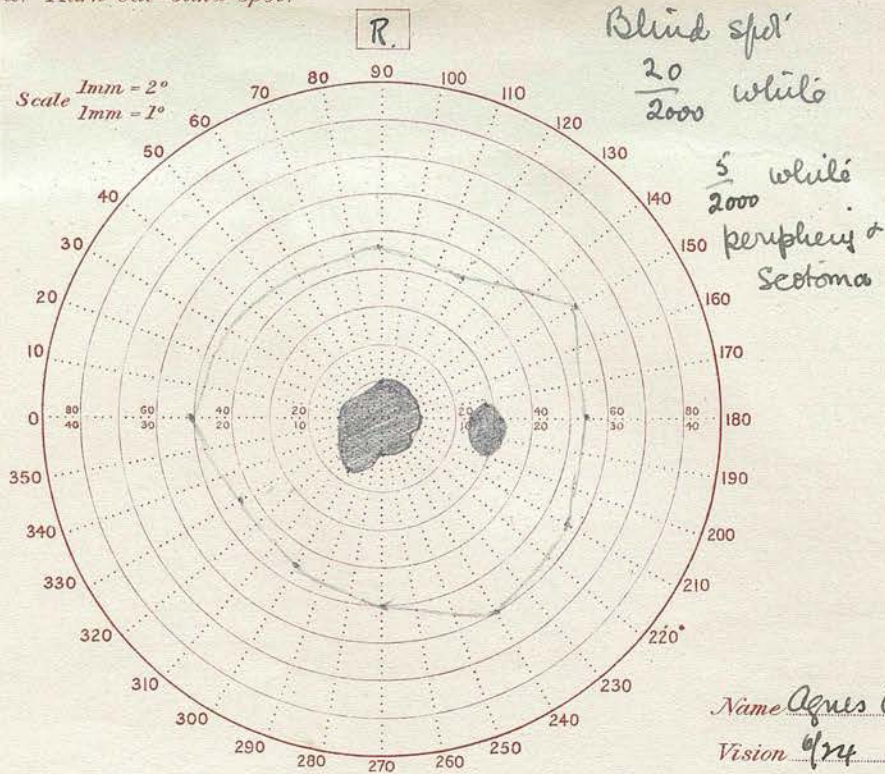
2.12.29. V. = C F at 8 in.

3.12.29. Ear, Nose and Throat - X-ray. Noth-
ing found.

Can read 1/60 in upper nasal field. Central
scotoma is more pronounced. Ocular pain most marked
on looking up.

7.12.29. Oedema still round disc. Scotoma
most marked down

d. Mark out blind spot.



Name Agnes Anthony

Vision 9/24

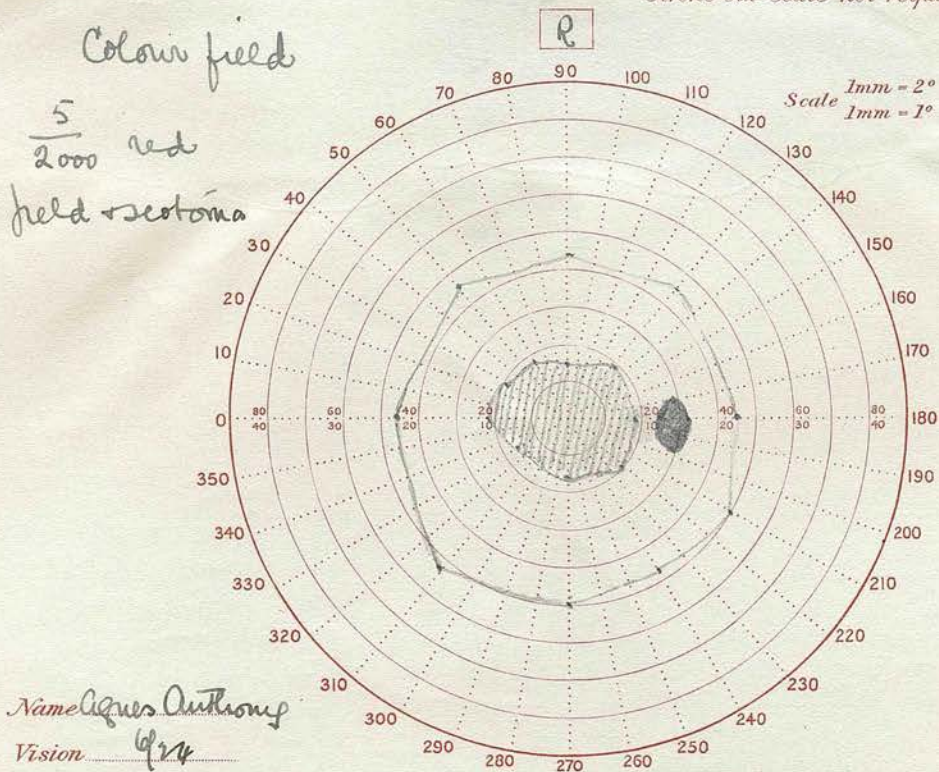
Date 21.2.30.

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Stroke out Scale not required

Colour field

5
2000 red
field & scotoma



Name Agnes Anthony

Vision 9/24

Date 21.2.30.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

8.12.29. V. 1/60 except centrally down.
10.12.29. V. 2/60.
11.12.29. V. 3/60.
12.12.29. V. 4/60.
13.12.29. V. 6/6. Less swelling n. head
 V. = 6/18. Scotoma below now absent.
14.12.29. Discharged.
21.12.29. V. = 6/6 V. 6/18 +
4.1.30. V. = 6/6 J 1. Rel. pallor of disc.
1.2.30. Four days ago sight of right eye suddenly became dim. Pain on movement of eye and tenderness on pressure, 4 days.

Central scotoma. Pupil does not hold. Contraction to light. Fundus normal.

Admitted.

2.2.30. R.V. = 6/60.
3.2.30. = .5/60.
4.2.30. = .5/60.
6.2.30. = .5/60. Some oedema upper edge of disc. Veins engorged.
7.2.30. .5/60.
8.2.30. 0.75/60.
9.2.30. 0.75/60.
10.2.30. 2/60.
11.2.30. 2/30. Field taken.
12.2.30. 2/60 +
14.2.30. 3/60 +

<u>15.2.30.</u>	3/60 +	
<u>17.2.30.</u>	3/60.	Oedema upper edge disc
		more marked and veins very swollen.
<u>18.2.30.</u>	6/60.	
<u>19.2.30.</u>	6/36.	
<u>20.2.30.</u>	6/36 +	
<u>21.2.30.</u>	6/24 (1)	Field taken.
<u>1.3.30.</u>	6/12 (4)	J 2.
<u>15.3.30.</u>	R.V. = 6/6 (-2)	J 1 L.V. = 6/6 ft. J 1.

Summary.

Typical case showing complete recovery.

Case VIII.

Jane Bruff, Domestic Servant, 30 N. Priestfield
Road. Age 34.

11.2.30. Complaint: One week ago had some
pain round about left eye. Three days ago, sight
of left eye affected, central objects being lost
first.

Examination: Pupil reacts only to a very
bright light and reaction is not held. Now has no
tenderness on pressure, and no pain on movement of
eyeball. Both discs are pale coloured, but not
without the bounds of the normal.

Knee jerks exaggerated.

No intention tremor.

Has been more tired during last two weeks.

$\frac{1}{2}$ c.r.

$\frac{1}{2}$ c.r.

R.V. $\bar{C} + 0.5 = 6/6$

L.V. = no P.L.

Admitted.

Neurological Examination negative.

Ear, Nose and Throat examination negative.

15.2.30. L.V. = No P.L.

16.2.30. L.V. = H.M. at 1 metre.

18.2.30. L.V. = Disc pale. H.M.

19.2.30. L.V. = H.M.

20.2.30. L.V. = H.M.

22.2.30. L.V. = H.M.

23.2.30. L.V. = Counts fingers at 1 metre.

$$\underline{26.2.30.} \quad \text{R.V. } \bar{c} \quad \frac{+0.5}{+0.5} = 6/6 \quad J \ 1. \\ 90^\circ$$

$$\text{L.V. } \bar{c} \quad +0.5 = 6/12 \ (2) \ J \ 10. \\ 90^\circ$$

$$\underline{27.2.30.} \quad \text{L.V. } \bar{c} \quad +0.5 = 6/9 \ (2) \ J \ 1. \\ 90^\circ$$

$$\underline{28.2.30.} \quad \text{L.V. } \bar{c} \quad +0.5 = 6/9 \ (2) \\ 90^\circ$$

$$\underline{1.3.30.} \quad \text{L.V. } \bar{c} \quad +0.5 = 6/9 \\ 90^\circ$$

Summary.

Typical case showing complete recovery.

Case IX.

Mrs Mary Greig, 61 Buccleuch Street. Age 32.

History: Lost sight of right eye on New Year's day. Sight now i.s.q.

Examination: No pupillary reaction, left eye.

Nearly 2 c.r.

$1\frac{1}{2}$ c.r.

R.V. \bar{c} 1.5 = 6/6

L.V. = H.M.

90°

Temporal

Neurological examination negative.

Ear, Nose and Throat examination negative.

14.2.30. L.V. = P.L.

15.2.30. L.V. = P.L.

18.2.30. Left disc shows slight pallor and the vessels on the disc are smaller than in the right.

L.V. = H.M. at $\frac{1}{2}$ m.

19.2.30. L.V. = H.M.

20.2.30. L.V. = H.M.

21.2.30. L.V. = H.M.

27.2.30. L.V. = F. at $\frac{1}{2}$ m.

28.2.30. L.V. = F. at $\frac{1}{2}$ m.

1.3.30. L.V. = F. at $\frac{1}{2}$ m.

Summary.

Uncommon type of case showing practically no recovery. Compare with Case IV.

Case X.

William West, 5 Elm Place. Age 30.

10.6.30. Complaint: Failing vision right eye.

History: Patient states that the vision of the right eye became poor four days ago and has continued to get worse. Patient is subject to colds.

Examination: There is pain on movement of the right eye, but no tenderness on pressure.

Right pupil reaction is very sluggish.

Left pupil reaction normal.

Teeth are in a very bad state.

R.V. = Hand movements. L.V. = 6/6.

11.6.30. Examination by physician with a view to disseminated sclerosis is entirely negative. Right vision still too bad to gain anything from perimetry. Central scotoma present for hand movements.

17.6.30. Examination by rhinologist negative.

X-ray negative.

Wassermann negative.

Teeth have been removed.

1.7.30. Sight remained poor for some time and then improved rapidly.

8.7.30. R.V. = 6/12.

Summary.

Typical case showing good recovery of vision.

DISSEMINATED SCLEROSIS.

The lesions of disseminated sclerosis may be found at any level in the visual pathway. They are, however, commonest in the optic nerve, less so in the chiasma, and rare in the tract.

The field changes are of two types:-

- (a) Gross central failure generally unassociated with other manifestations of disseminated sclerosis.
- (b) Slight defects of central vision in patients who show other signs of disseminated sclerosis.

(a) Patients belonging to the first group show unilateral failure of central vision and the field changes are those associated with acute retrobulbar neuritis.

This failure comes on very rapidly and is often of great severity. Recovery in the majority of cases is also rapid and very good, but in a few cases the end result is bad. This type of visual loss is not commonly associated with other manifestations of disseminated sclerosis.

The field changes consist of a dense or absolute central scotoma surrounded by an area of relative

scotoma and finally with a ring of unaffected peripheral field. If the central defect is very great, it may break through to the periphery.

(b) Patients belonging to the second group show multiple small relative central or paracentral scotomata which are vague and indeterminate. They are frequently bilateral and are associated with other manifestations of disseminated sclerosis.

The above two groups of field changes occur when the site of the lesion is in the retrobulbar portion of the optic nerve. If, however, the lesion is situated at the termination of the nerve, unilateral hemianopic scotomata are produced. If the lesion is still higher, bitemporal or homonymous hemianopic scotomatous defects occur.

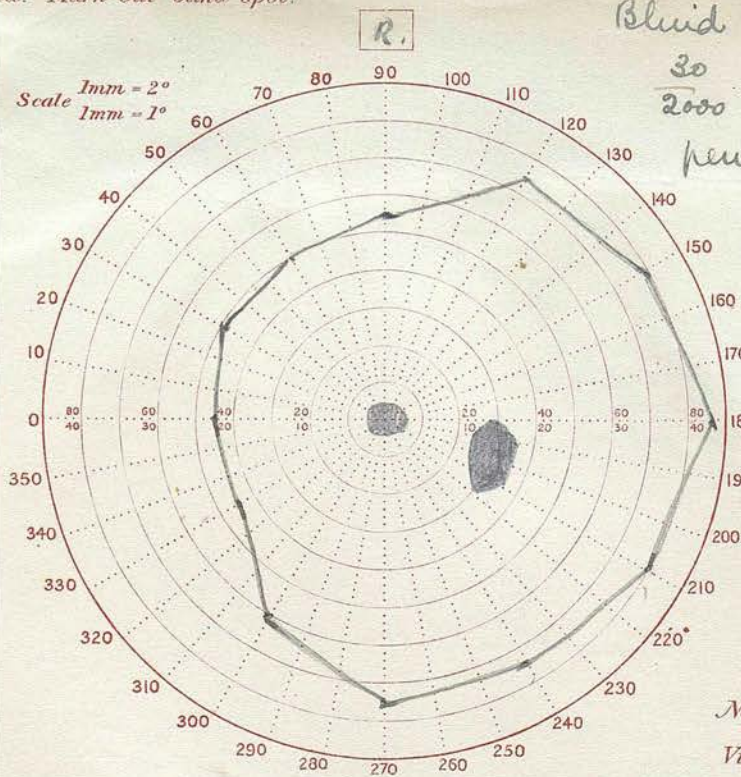
CASES.

Series of 10 consecutive cases of disseminated sclerosis, chosen without any regard to field defects, gave the following findings:-

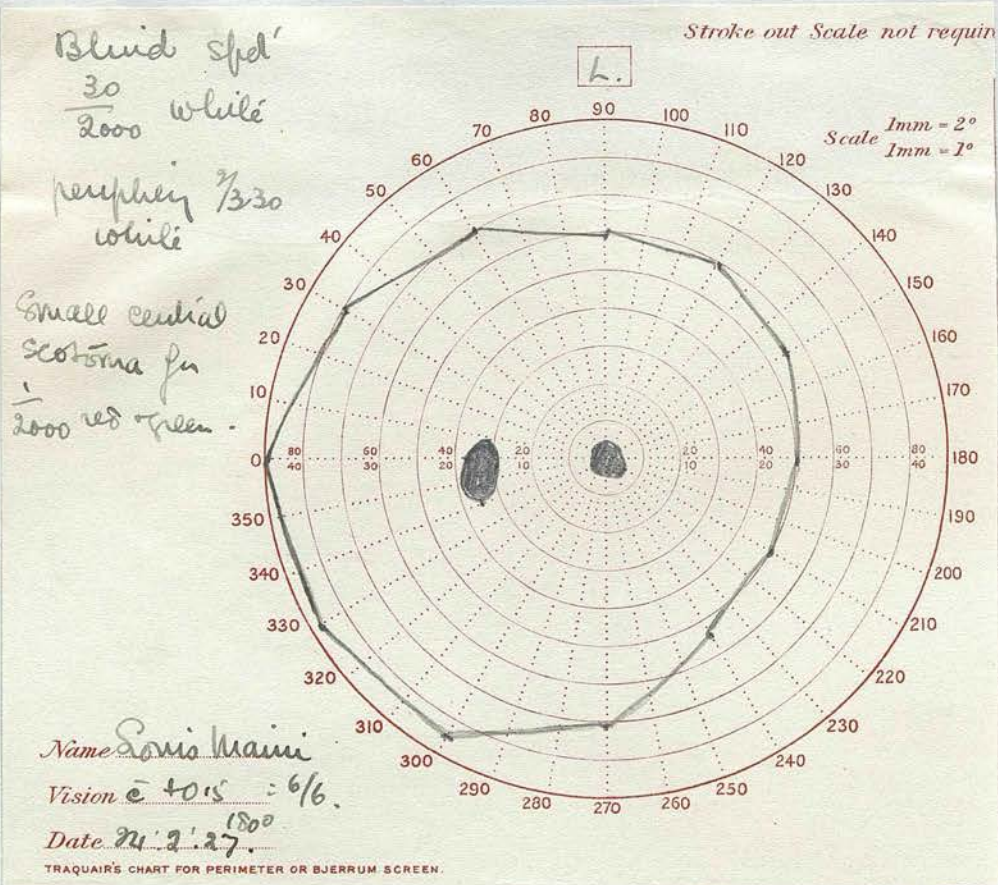
CASE II DISSEMINATED SCLEROSIS

LOUIS MAINI

d. Mark out blind spot.



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TRAQUIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Case 1.

16.2.27. Man, aged 29. Never had any failure of vision: only ocular sign, nystagmus.

R.V. = 6/6 + J 1. L.V. = 6/6 + J 1.

Field examined with 2/330 white, and with 1/2000 white, red and green test objects, showed no abnormality.

Case 2. Louis Maini.

24.2.27. Man, aged 18. Complained of diplopia, in July 1926, of two weeks' duration. Nystagmus present. No complaint of failure of vision.

R.V. $\bar{c} + 0.5 = 6/6$ J.1. 180° L.V. $\bar{c} + 0.5 = 6/6$ J 1. 180°

Field changes. Small central scotoma present in each eye for 1/2000 red and green.

These scotomata were vague with indistinct margins, but patient was fairly definite as to their presence. This case therefore belongs to Group (2).

Chart of fields attached.

Case 3.

25.2.27. Man, aged 27. No complaint of failure of vision. No ocular signs or symptoms. No field defect found with 2/330 white or 1/2000 red or green.

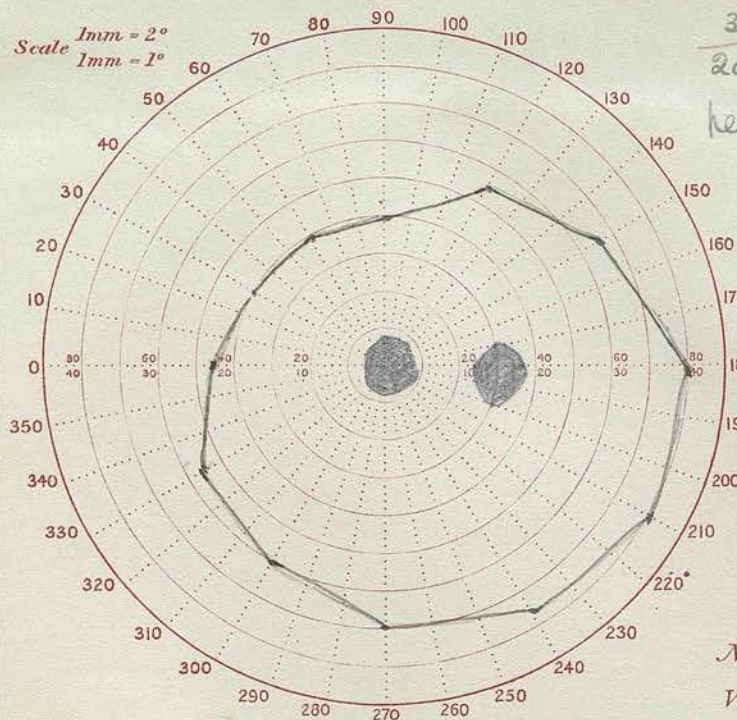
$$R.V. = 6/6 + J \text{ l.} \quad L.V. = 6/6 + J \text{ l.}$$

MRS COLQUHOUN

d. Mark out blind spot

R

Blind spot

Scale 1mm = 2°
1mm = 1°30
2000 white

periphery

2
330 whiteSmall central
Scotoma for1
2000 red &
green.

Name Mrs Colquhoun

Vision 6/6 - 0.5 90°

Date 28.2.27

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Blind spot

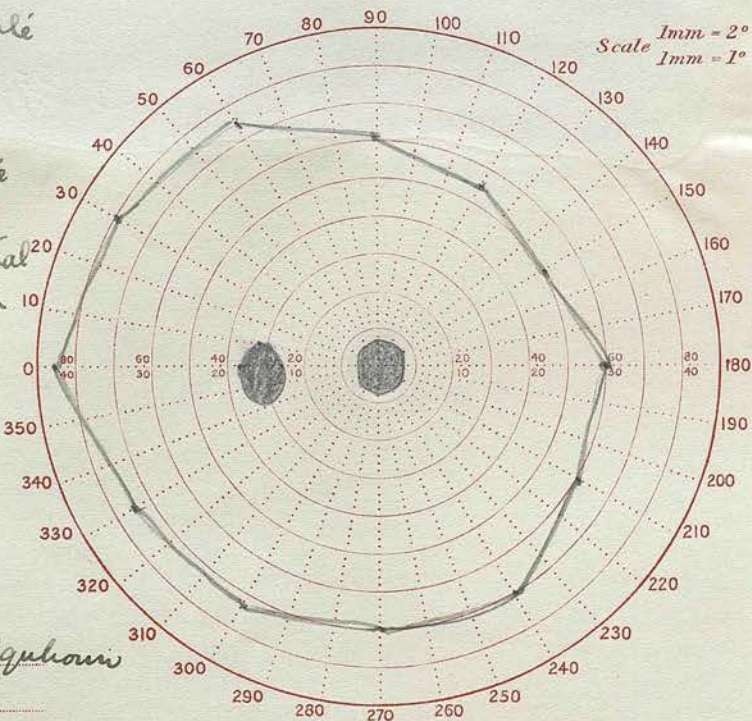
30
2000 white

periphery

2
330 whiteSmall central
Scotoma for1
2000 red &
green.

Stroke out Scale not require

L

Scale 1mm = 2°
1mm = 1°

Name Mrs Colquhoun

Vision 6/6 -

Date 28.2.27

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Case 4. Mrs Colquhoun.

28.2.27. Woman, aged 43. Only complaint regarding vision is difficulty with reading, which is obviously due to presbyopia. No diplopia or nystagmus.

$$\text{R.V. } \bar{c} + 0.5 = \frac{6}{6} \quad 100^\circ$$

$$\text{L.V. } \bar{c} + 0.5 = \frac{6}{6} \quad 90^\circ$$

$$\bar{c} \frac{+1.}{+0.5} = J 1 \quad 100^\circ$$

$$\bar{c} \frac{+1.}{+0.5} = J 1 \quad 90^\circ$$

Field changes. Field with 2/330 showed no abnormality. Small central scotoma in each eye for 1/2000 red and green.

This case also belongs to Group (2).

Chart of fields attached.

Case 5.

Man, aged 54. Two years previously had diplopia. Duration one week. No ocular symptoms or signs now present. No field defect with 2/330 white, 1/2000 red, green or white.

$$\text{R.V.} = \frac{6}{6}.$$

$$\text{L.V.} = \frac{6}{6}$$

$$\bar{c} + 2 = J 1$$

$$\bar{c} + 2 = J 1$$

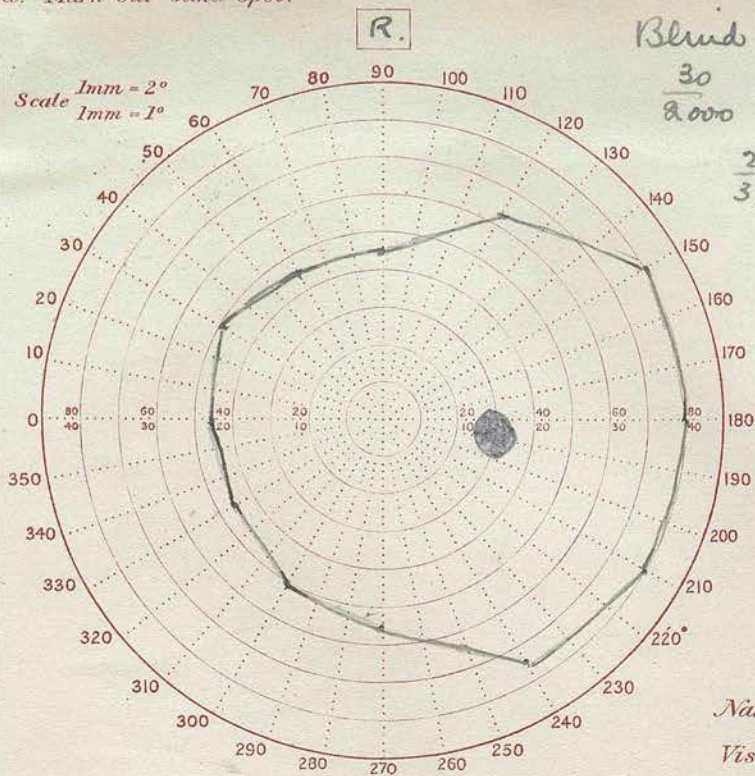
Case 6. Mr Bruce.

16.3.27. Man, aged 38. (Group 1)

Sixteen years previously, sight of right eye failed almost entirely and then returned.

BRUCE

ed. Mark out blind spot.

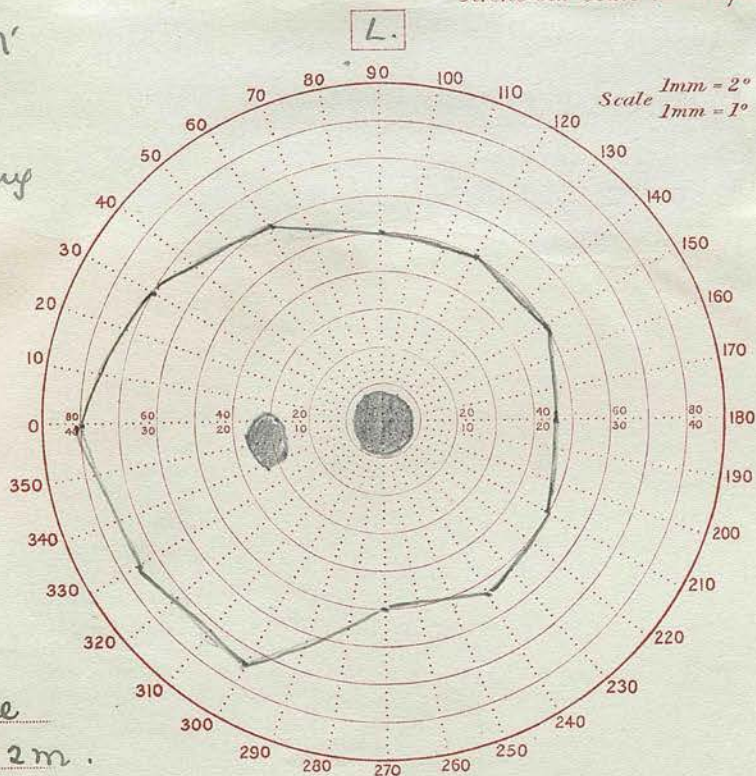


THE DARIEN PRESS, 5 BRISTOL PLACE, EDINBURGH.

Stroke out Scale not required

Blind spot
 $\frac{30}{2000}$ white
 $\frac{2}{330}$ periphery

Scotoma
 $\frac{15'}{2000}$



TRAQUAIR'S CHART FOR PERIMETER OR SJERRUM SCREEN.

Fifteen years previously, same condition occurred in left eye.

Right pupil reacts sluggishly to light; other pupillary reactions normal. Convergence poor, nystagmus to left.

Left divergent squint.

R.V. = 6/24 with - 0.5 = 6/18 L.V. F at $1\frac{1}{2}$ metres
c 5. c 20.

Left field shows presence of central scotoma.
Chart of fields attached.

Case 7.

19.3.27. Man, aged 28. No history of loss of vision. No diplopia. Nystagmus so bad that fields could not be taken, but there was nothing to indicate presence of central scotoma.

$$\text{R.V. } \frac{\bar{c} + 0.5}{150} = 6/6 \quad \text{J l.} \quad \text{L.V. } \frac{\bar{c} - 0.5}{180} = 6/6 \quad \text{J l.}$$

Case 8.

Man, aged 42. No ocular complaint. No field defects.

R.V. = 6/4 J l. L.V. = 6/4 J l.

Case 9.

27.4.27. Man, aged 38. (Group 2)

In 1914 lost sight of left eye. Four or five

months later, right eye failed, but never so badly.
Both improved.

In 1924, diplopia: not now present.

R.V. = 6/9 J 1. L.V. = 6/12 J 4.

Central scotoma present in both fields.

Case 10.

10.5.27. Man, aged 43. No ocular complaint - symptom or sign.

$$\text{R.V. } \bar{c} - 0.5 = 6/6 \quad \text{J } 1 \quad \text{L.V. } \bar{c} + 0.75 = 6/6 \quad \text{J } 1$$

In a survey of the foregoing ten cases, numbers 1, 3, 5, 7, 8 and 10 may be dismissed as bearing no close relationship to the subject of this thesis.

Numbers 2 and 4 belong to the second group of cases. They both show small central scotomata for a very tiny coloured object. These scotomata have uncertain edges and do not depress vision, as recorded by Snellen or Jaeger types, sufficiently to bring them to the notice of the patient.

Numbers 6 and 9 belong to the first group of cases. Both give histories of having lost the sight of first one eye and then the other several years before. Vision returned, but in neither case to normal. Obviously the patients suffered from attacks of acute retrobulbar neuritis and at

periods varying from thirteen to sixteen years afterwards, other symptoms of disseminated sclerosis appeared. They both still show the presence of central scotoma.

It is of interest to note that an article on the relationship between retrobulbar neuritis and disseminated sclerosis appeared in the Lancet, July 5th, 1930. In it the author (C. P. Symonds) states that in a series of 139 cases of disseminated sclerosis, a history of an attack of retrobulbar neuritis was obtained in 28%, and in 15% of cases it was the first symptom. He also gives notes of 3 cases in which the latent period between the attack of retrobulbar neuritis and further symptoms of disseminated sclerosis was as long as 17, 10 and 18 years.

NASAL ACCESSORY SINUS DISEASE.

Nasal accessory sinus disease is not a common cause of Retrobulbar Neuritis, but when it does occur, the affections are obvious, the sphenoidal and ethmoidal cells being the sinuses involved.

Cases of retrobulbar neuritis due to sinus disease fall naturally into two groups:-

1. Those which are seen first by the ophthalmologist, who finds gross field changes. These cases he desires to be examined by the rhinologist, and if obvious sinus disease is discovered, it can be taken as the cause.
2. Those seen first by the rhinologist, who has the cases examined by the ophthalmologist following his diagnosis of obvious or "latent" sinus infection.

Cases which have an obvious affection of the sinuses do give rise to an acute retrobulbar neuritis with typical field changes. Unfortunately I have not as yet come across an illustrative case, and rather than use cases of other observers, have contented myself with descriptions obtained from the study of the literature.

The scotoma is usually pericentral and may show fluctuations, or the nerve condition may progress to atrophy. It does not follow the typical course of recovery as seen in retrobulbar neuritis from other causes. All cases of retrobulbar neuritis should have the nasal sinuses examined as a matter of routine, and if obvious disease is found, its treatment will be the treatment of the retrobulbar neuritis.

When we come to deal with the so-called "latent" sinus disease, we then find a very different group of cases, a group over which there has been a great deal of argument and controversy. Much work has been done on the rhinogenic origin of retrobulbar neuritis both in this country and abroad, and many and varied are the opinions thereon.

Commencing in March 1924 and continuing thereafter for two years, a joint investigation of this condition by oculists and rhinologists was set afoot in Edinburgh. In this investigation I had the privilege of playing a small part and since then have also studied a large number of cases in my own clinics and practice.

A series of 28 cases of retrobulbar neuritis which were examined in the Edinburgh investigation gave almost entirely negative nasal symptoms and signs, and in no case was evidence found of

suppurative sinus disease.

The examination of the fields of visions in cases of suspected sinus disease yields in the hands of different observers as many different results. Each observer finds one particular sign in his series of cases, e.g. enlargement of blind spot, and to this sign he adheres. This lack of consistency points to the findings being a product of the oculist's brain rather than the patient's eye.

To check these statements, I examined in 1926 a consecutive series of 25-30 cases of patients who were about to undergo Sluder's operation for suspected sinus disease. In not one single case did I find any departure from the physiological limits of the field, any enlargement of the blind spot or the presence of any scotomata, and this although the test objects used were very small.

In very many cases of acute retrobulbar neuritis the ethmoid and sphenoid sinuses have been opened in the hopes that some lesion might be found, if not macroscopically then microscopically. In many cases an absolutely normal mucous membrane and in a few a congested mucous membrane was found. There seemed to be no connection between the disease and the finding. The majority of cases got better, but as they also get better without any treatment, one simply cannot accept blindly the statement that the

cure was the result of the operation - post hoc, ergo propter hoc. In one case (Adams, bilateral chronic retrobulbar neuritis) where congested mucous membrane was found and where if anywhere help might have been expected, the vision went from bad to worse.

Meller and Hirsch are inclined towards the view that latent sinus disease is an actuality and that rhinological treatment, drastic or otherwise, is a *sine qua non*.

Davies, reporting on a series of 54 cases of retrobulbar neuritis which were examined by him rhinologically, states that only in 4 out of these 54 cases was the neuritis produced by nasal suppuration and in them the disease was obvious and extensive. He further says that an improvement in the sight after the sphenoid and ethmoid have been opened and when little or no disease was found does not conclusively prove that the neuritis was caused by the nasal condition.

My own opinion is that unless disease of the nasal accessory sinuses is obvious, it does not cause retrobulbar neuritis.

CHRONIC RETROBULBAR NEURITIS.

If we exclude tobacco amblyopia, chronic retrobulbar neuritis is a rare condition.

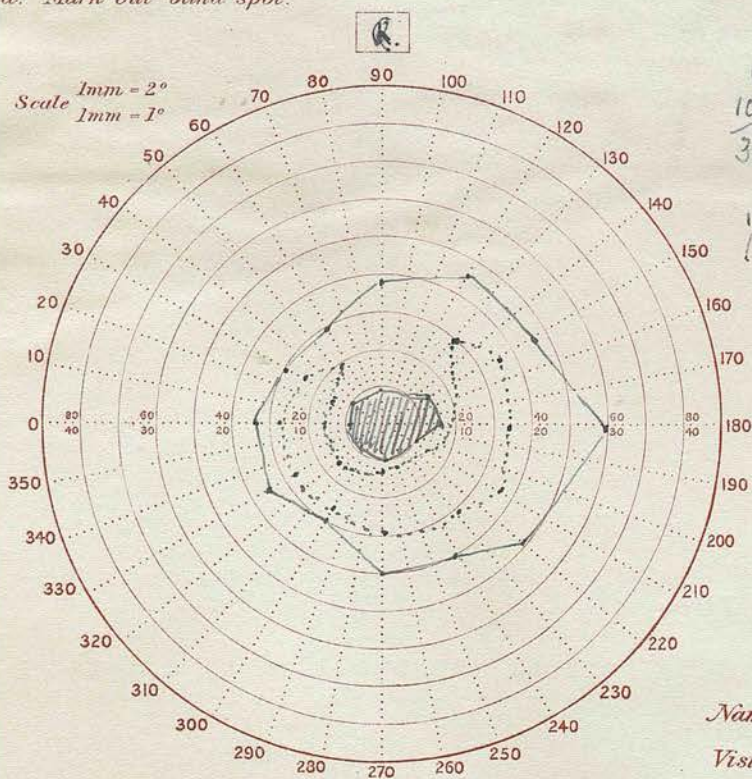
Certain cases of retrobulbar neuritis run a much slower course than the typical acute form. These are usually bilateral, show fundus changes, i.e. optic neuritis in early stages, do not recover so quickly or so well, and often finish with poor vision and a certain amount of atrophy of optic discs.

CASE OF CHRONIC RETROBULBAR NEURITIS

opp

ROBERT ADAMS

d. Mark out blind spot.



while objects
10
330 peripheral
scotoma

1/330 showing
breaking
through

Name Robt. Adams

Vision R. 7 at 3 ft.

Date 14/1/29

THE DARLEN PRESS, 5 BRISTOL PLACE, EDINBURGH.

Robert Adams, schoolboy, aged 11.

27.6.24. Complaint: Failing vision, both eyes.

History: Patient gave a history of failing vision in both eyes, which he first noticed two weeks previously.

Previous illnesses - Whooping cough and measles, age 5.

No other illnesses.

Examination: No refractive error. Both pupils had typical "retrobulbar" reaction, i.e. they reacted to light, but the reaction was not held.

Both discs showed neuritic changes, with swelling.

R.V. = 1/80. L.V. = 1/80.

At this time patient was examined by a physician and a radiologist, whose reports were negative, and by a rhinologist, who also could find no obvious signs of involvement of nose, etc.

7.7.24. - R.V. 1/80. L.V. 1/80.

Neuritis and swelling still very pronounced.

About this date a Sluder operation was performed by the rhinologist in the hopes that some infection might be found in the sphenoid or ethmoid air cells. No pus was found, but the mucous membrane was very congested. This was cleared out, but

with no improvement in the vision.

22.8.24. Still some swelling to inner side R. disc. Atrophic changes showing in both discs.

19.9.24. Atrophy, R. disc. Slight atrophy L. disc. R.V. = F. at 2 feet. L.V. = F. at 2 feet.

26.9.24. I.s.q.

7.10.24. R.V. = F. at 2-3 feet. L.V. = F. at 2-3 feet. Fundi the same.

Examination five years later:

11.10.29. Pronounced atrophy of both discs.

R.V. = F. at 1-2 feet L.V. = 6/9 J 1.

Apart from the congested mucous membrane found in the sphenoid and ethmoid air cells, no other cause of the retrobulbar neuritis was found. Very little recovery took place at the time, but on examination five years later, vision was found to have recovered very considerably in the left eye. Compare notes on accessory sinuses.

(2) TOTAL TRANSVERSE ACUTE RETROBULBAR NEURITIS.

This condition is comparable to a very severe case of acute axial neuritis which has involved the entire extent of the nerve.

It is usually bilateral, and the failure of vision is rapid and severe.

Often no cause of the neuritis can be found, but it may follow influenza and other febrile conditions.

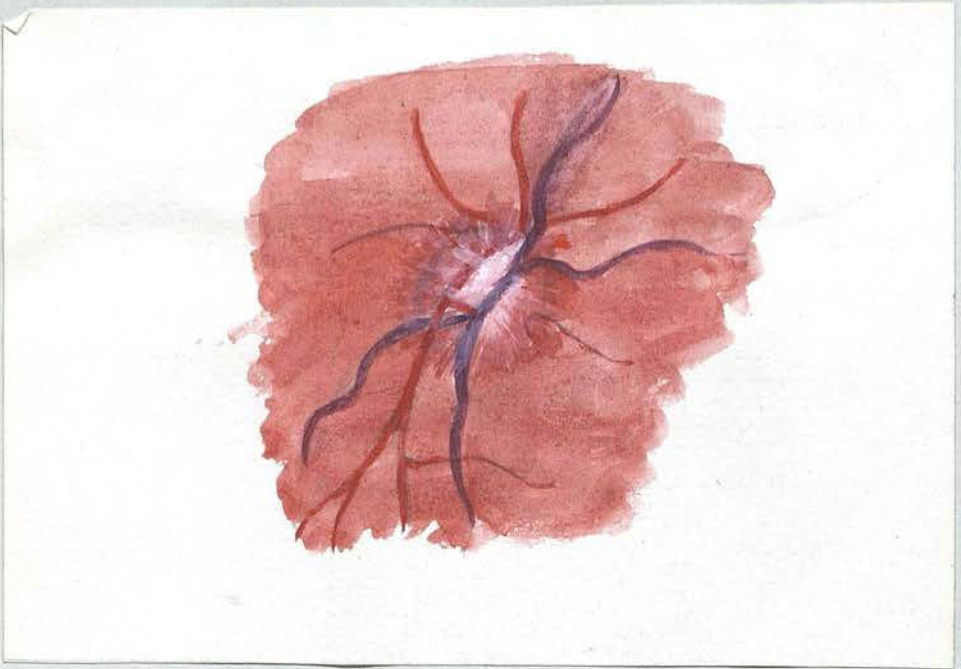
One interesting form is definitely associated with myelitis, the spinal symptoms of which appear at a later date.

Rapidly occurring bilateral blindness is sometimes found in children. On examination, both discs are swollen and show evidence of neuritis. This may be a form of that which is associated with myelitis, but without the spinal symptoms.

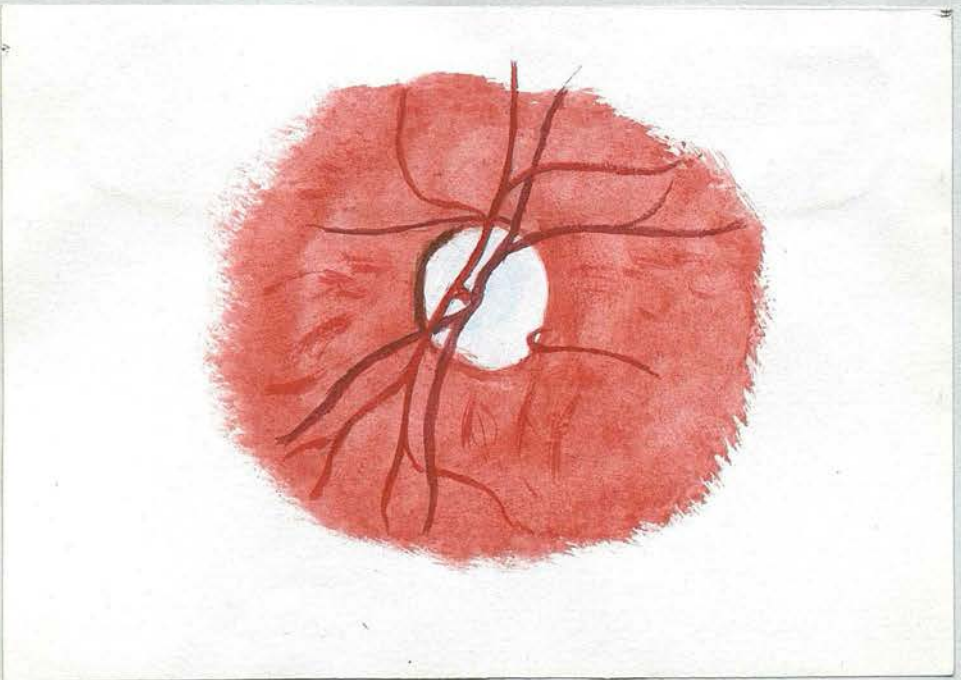
Owing to the fact that failure of vision is so rapid, and that frequently the patients are children, charts of field defects are difficult to obtain. Sometimes, however, particularly during recovery, a central scotoma may be demonstrated with a large object.

The only original case of this kind that I have so far come across occurred in a little girl of five years. It was not associated with myelitis, and no cause of the neuritis was ever found.

ISOBEL NELSON



OPTIC NEURITIS 1925



OPTIC ATROPHY 1929

Case of Acute Bilateral Retrobulbar Neuritis.

Isabel Nelson, aged 5, at school.

2.6.27. On Thursday, June 2nd, the teacher at school noticed that patient was not seeing as well as usual in the morning, and by the afternoon the child appeared to be quite blind.

Previous history: Only illnesses, measles and whooping cough. Intermittent earache, but no history of discharging ear obtained. During the last 2-3 weeks, child has been irritable and peevish and inclined to sleep more than usual.

On Examination, both pupils dilated and fixed.

Double optic neuritis. Both discs were greatly swollen and veins congested. Apparently no P.L. in either eye.

3.6.27. Aurist's examination:- Nose, sinuses and throat negative. Left ear, wax: after syringing, tympanic membrane totally destroyed. Right ear also contains wax.

Neurologist's examination: Negative.

Cerebrospinal fluid not under pressure and clear.

X-ray examination: Negative.

3.6.27. Left pupil reacts and then dilates.

V. - P.L. Discs 1.s.q.

4.6.27. Left pupil reacts quite briskly when

light is thrown on periphery. Veins even more swollen.

5.6.27. No reaction to light. I.s.q.

Brighter.

6.6.27. I.s.q.

7.6.27. I.s.q.

8.6.27. Left pupil reacting. Admits P.L. but doubtful.

11.6.27. No reaction. No vision.

14.6.27. Discs becoming atrophic.

20.6.27. Atrophy much more pronounced.

24.6.27. Main evidence that vision was recovering. Two eyes not tested separately.

26.6.27. Able to pick up small coins on theatre floor.

27.6.27. Counting fingers at $\frac{1}{2}$ metre in left eye. Right eye appears to have no sight.

3.7.27. Vision also in right eye. Atrophy of both discs, worse in left. Child has been able to run about the Ward and play with the others; also to recognise and pick up objects on the floor.

26.7.27. Discharged.

9.8.27. Both pupils react briskly. Discs pale, but arteries not unduly small.

Very little evidence of previous oedema.

Two years later. 9.10.29. Aged 8.

Pupils react briskly to light.

No refractive error.

Both discs very pale, but vessels not small and very little sign of previous oedema. One or two pigmented patches on the choroid of right fundus.

R.V. - 6/12 J 1. L.V. = 6/18 J 1 (halting).

An attempt was made to chart the fields of vision, but child was unable to fix properly. The only facts pointing to the presence of central scotoma were volunteered by a relative who stated that the child seldom looked straight at her, but usually a little above her head, and if picking up an object, would look to one side. These observations were confirmed by me.

From the foregoing notes it will be seen that all attempts at discovering the cause of this intense inflammation of the optic nerves was a failure. The only feasible suggestion is that it is an example of a case closely allied to those associated with myelitis in which the spinal symptoms are absent.

The child is now perfectly well, able to see sufficiently to do her school work and play with other children, and is very smart with her replies during subjective testing.

LEBER'S DISEASE, also called Hereditary Optic Atrophy, or Hereditary Optic Neuritis.

This is a hereditary disease which takes the form of a subacute retrobulbar neuritis, affecting both eyes, either at the same time or with a short interval between. It occurs usually in many members of the same family, mostly males, and is transmitted through the females, in whom it is rare. Isolated cases, with no family history, also occur. The disease commences in one of two periods, either during adolescence, or later about 40 to 50.

History and Course: The disease is characterised by a failure of sight coming on rapidly, usually bilateral and getting worse for a few months. After this, improvement sets in, but perfect vision is never regained, nor does total blindness occur.

Ophthalmoscopic Appearances: Frequently the fundi appear normal, and later, pallor of the disc may develop without any previous neuritis.

In other cases, a mild degree of change in the nerve head occurs, which takes the form of slight blurring of the disc margin, but haemorrhages or exudates do not occur.

Field changes: The characteristic field change is the presence of a central scotoma. This is a true central or pericentral scotoma. It varies in

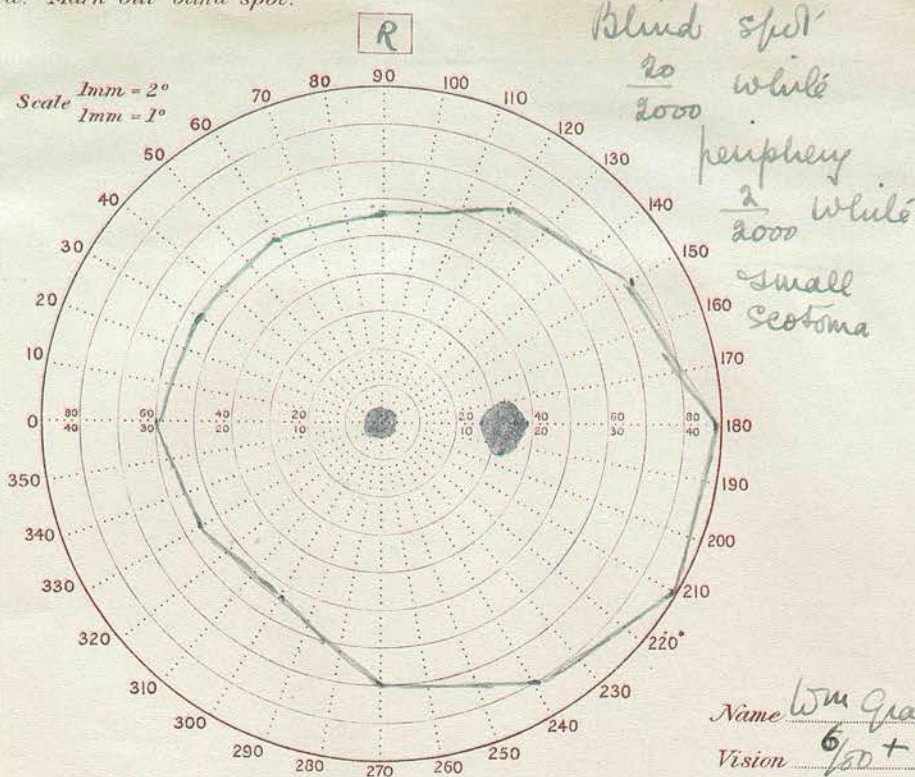
size and shape, but is usually roughly circular. It varies also in intensity; a scotoma for a larger object is found within that for a smaller. The very centre of the scotoma may be absolute. In one case a paracentral scotoma as well as a pericentral was found for the white object 2/2000.

Summary.

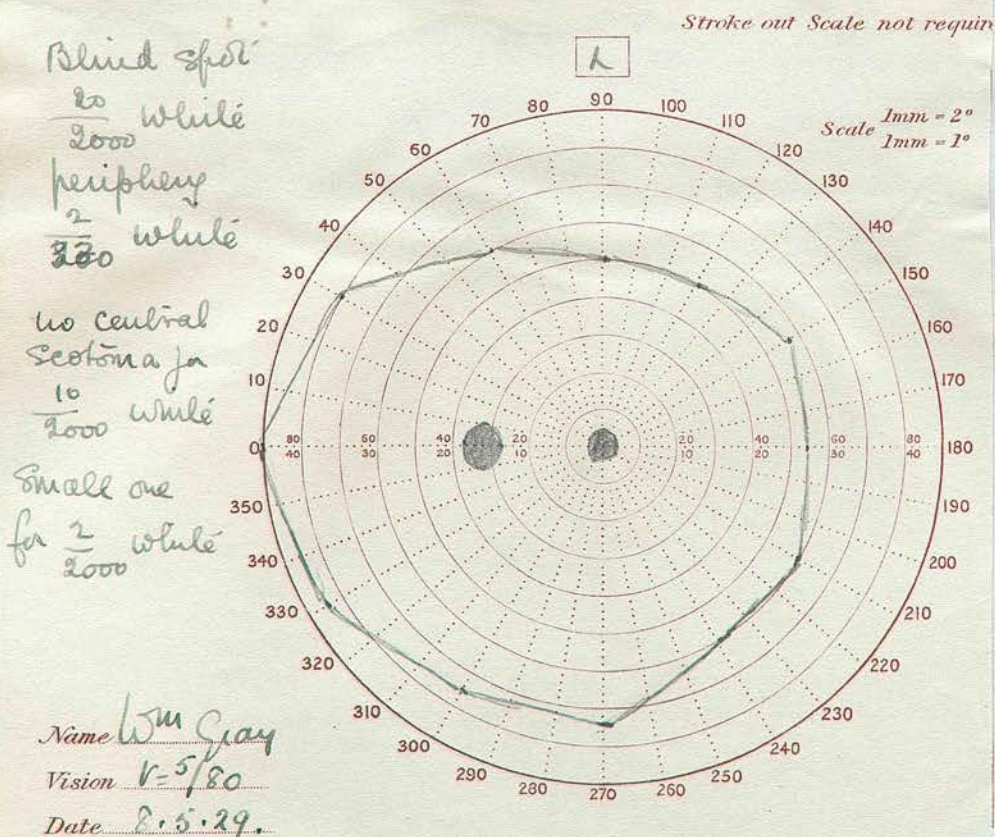
The field picture obtained indicates that the lesion is situated below the chiasma, presumably in the nerve. The changes are similar to those obtained in some forms of bilateral retrobulbar neuritis, and for purposes of diagnosis the whole clinical picture must be taken into consideration. The field changes do, however, differentiate this condition from tobacco amblyopia (scotoma, centro-caecal) and from chiasmal condition (scotoma hemianopic).

WILLIAM GRAY

d. Mark out blind spot.



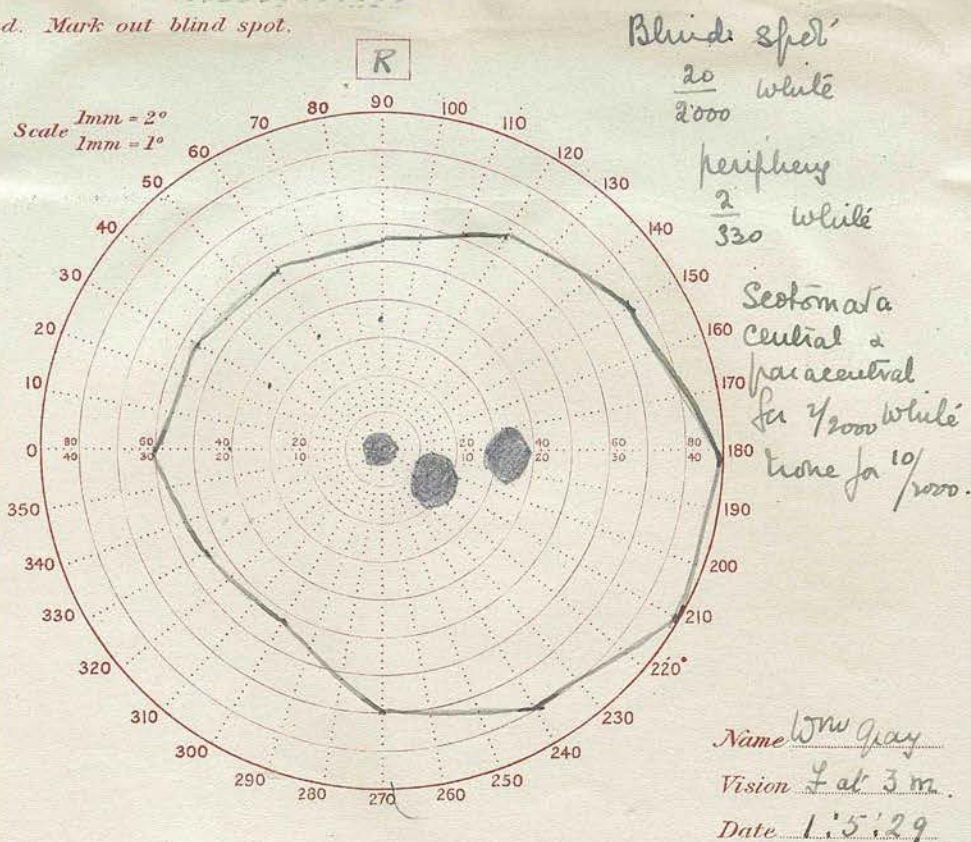
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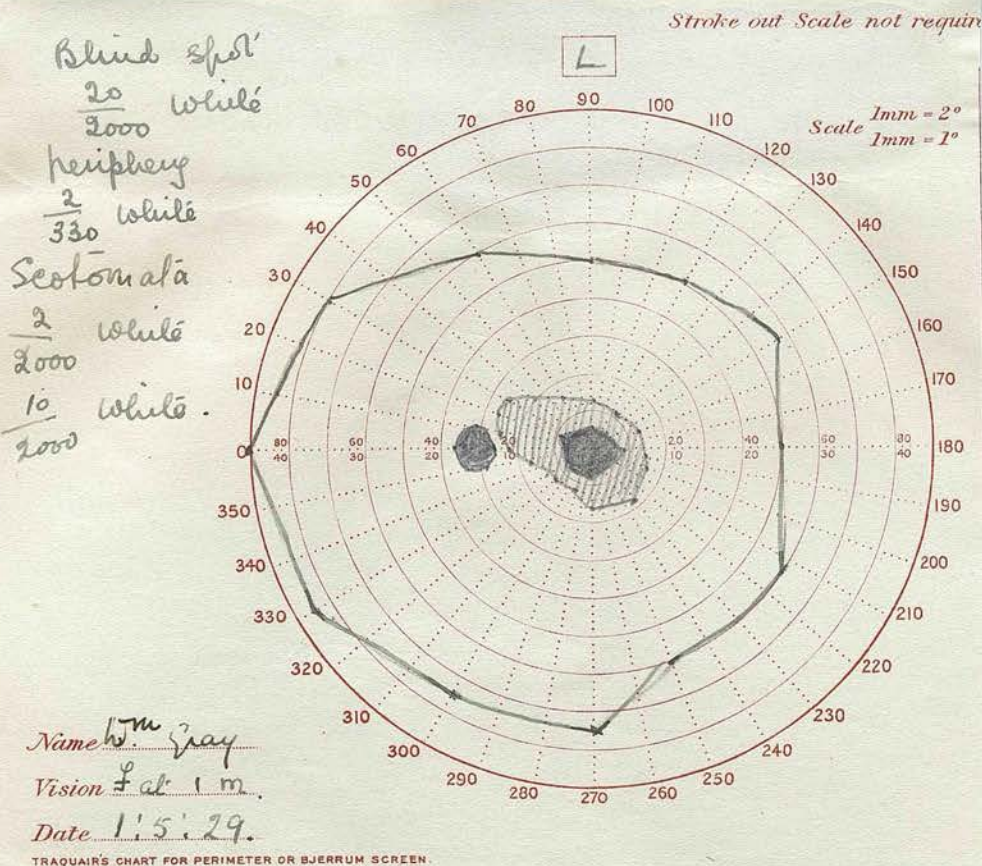
TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

WILLIAM GRAY

d. Mark out blind spot.



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TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

Case of Leber's Disease.

William Gray. Age 23. Miner.

11.4.29. Complaint: "Failure of vision in both eyes."

History: Patient states he noticed vision of both eyes began to fail about one month previously. No hereditary history.

Examination: Fundi healthy.

Central failure in both fields of vision.

R.V. = 5/80. L.V. = 2/80.

22.4.29. X-ray examination negative.

1.5.29. Pupils react. R. pupil larger than L.

R.V. - F. at 3 m. C 12. L.V. - F. at Im. C 12.

Fields taken - Periphery 2/330, centre 10/2000, 2/2000 white.

Case of Subacute Bilateral
Retrobulbar Neuritis.

David Low, Farmer, Broomfield, Ellon, Aberdeenshire. Age 31.

20.8.30. Complaint: Failure of vision of right eye.

History: On August 17th, 1930, patient had a bad cold, with frontal headache, and a temperature which rose to 101° on August 18th. On August 19th, rapid profound loss of vision in right eye took place, accompanied by pain on movement.

20.8.30. Called at Collieston, but was not seen by me.

21.8.30. Seen by Dr Soutar in Aberdeen, who diagnosed retrobulbar neuritis. On this day the left eye became affected in a similar manner.

24.8.30. Seen by me.

Right pupillary reaction very poor and not sustained.

Right vision = Finger counting close.

Right disc margin shows no blurring, but veins are congested.

Left pupillary reaction brisker than right, but not well sustained.

Left vision = Finger counting 1-2 metres.

Left disc margin blurred upper nasal portion

and veins very congested. Central scotoma present in both eyes.

30.8.30. Patient's general condition much better.

Both pupils react more briskly.

Congestion of retinal veins less pronounced, as also is blurring of disc margin in left eye.

R.V. = F. at 3 m. L.V. = F. at 6 m.

Owing to the fact that this patient was seen in a farmhouse in the country, it was impossible to obtain anything but the very roughest record of the field of vision. There was, however, no doubt that an extensive and dense central scotoma existed in each eye.

22/9/30 R.V. 6/9 L.V. 6/18

Query Leber's Disease

CONCLUSIONS ON RETROBULBAR NEURITIS.Pathology.

Acute retrobulbar neuritis is a fairly common disease and its cause while discoverable in a certain percentage of cases is in many more obscure. Forty per cent. by some observers is given as the figure for retrobulbar neuritis to occur as a very early symptom of disseminated sclerosis. It is only years afterwards, however, that in many cases we are able to confirm the diagnosis of the etiology by the development of further symptoms and signs of disseminated sclerosis. While actually examining the case one can only say that at the time no obvious cause was found, and the case may later become one of disseminated sclerosis. Time proves the diagnosis correct in many instances, and in others, no cause is ever found. The optic nerve is a very favourite site of the lesion of disseminated sclerosis. Looking at this condition from the reverse point of view, cases of disseminated sclerosis in many instances give a history of attacks of loss of vision many years before, and in some cases post neuritic atrophy or a persistent central scotoma confirms the patient's statement. In others, the presence of a small central scotoma is found on examination, and yet in others, no eye symptoms or signs are found. There are cases attached which illustrate these points.

A certain percentage, but very small, are without doubt due to demonstrable disease of the nasal accessory sinuses, and of these the posterior ethmoid cells and sphenoidal sinus are those chiefly affected. When the ophthalmologist has found pronounced field changes and the rhinologist pronounced sinus disease, it is only feasible to consider these as effect and cause.

There is, however, another group of cases which have always provided a good ground for argument, and these are the cases of so called "latent" sinus disease. For a fuller description, see a previous paragraph entitled "Nasal Accessory Sinus Disease".

Bilateral cases are more uncommon and are often definitely associated with some general condition, as myelitis,

Also in a class by itself we must put Leber's Disease, which is a definite entity showing all the typical signs of a subacute bilateral retrobulbar neuritis followed by atrophy.

It follows from the foregoing that these cases can be divided into three groups:-

1. Cases in which can be shown an obvious definite cause, e.g. disseminated sclerosis, Leber's disease, etc.

2. Cases in which the cause is dubious.

Among these there will be found the

adherents of "latent" sinus disease and septic teeth. Those of us who feel we cannot be so dogmatic will question these causes.

3. Cases in which nobody can honestly say they have found a cause.

Clinical Significance.

Perimetry is of great value in the diagnosis of retrobulbar neuritis. As in tobacco amblyopia, the refraction, media and fundi of a case may all be normal and the diagnosis must then be made from the field changes combined with the history. The typical field defect in these cases is a central scotoma. This time it is truly central or pericentral, and not as in the case of tobacco amblyopia, centro-caecal.

In the ordinary acute retrobulbar neuritis, the scotoma becomes denser until the climax of the condition is reached when it fades away and finally disappears leaving no trace.

In cases which show changes in the optic disc, the field defects serve as a means of diagnosing the condition from papilloedema, in which there is no central failure.

In certain chronic cases and in Leber's disease

central scotoma persists to a more or less extent,
depending on the amount of permanent damage done.

GROUP III.THE CHIASMA.

In the case of the optic nerve, we have seen that while pressure, vascular lesions and injury are not unknown, the commonest lesions affecting the nerve are intoxications and inflammations. Passing back to the chiasma, however, pressure becomes the most outstanding lesion, and this is seen par excellence in the case of pituitary tumours.

The characteristic field change which is found as a result of interference in the chiasma is a bi-temporal hemianopia, of which the two main types are scotomatous and non-scotomatous.

The progress of the scotoma is very similar to that of the general field.

In the earliest stages of field change as a result of pressure on the chiasma, a flattening or indentation of the upper temporal quadrant occurs. In certain cases this defect is only demonstrable with a 1/2000 white. As this defect increases, at the same time a scotoma appears in the apex of the upper temporal quadrant, reaching to the vertical

meridian on the one side and to the horizontal on the other. This scotoma enlarges in an upward and outward direction, and meeting the downgrowing peripheral indentation, breaks through to the periphery.

In the next stage the scotoma travels down into the apex of the lower temporal quadrant. The lower quadrant then fails at the periphery and grows up to meet the scotoma which again breaks through, sometimes leaving an isolated patch of field called the temporal island. At this stage a bitemporal hemianopia is formed.

Next the lower nasal field begins to fail and the scotoma passes into the apical part of the lower nasal quadrant, and finally the upper nasal field fails and the scotoma passes into the apical portion of the upper nasal quadrant.

The scotoma follows the progress of the periphery of the field - first the upper temporal quadrant, then lower temporal, lower nasal, and lastly upper nasal - clockwise in the right eye and counter-clockwise in the left.

It is often possible to demonstrate the presence of the scotoma in the apex of every quadrant and at different stages in each.

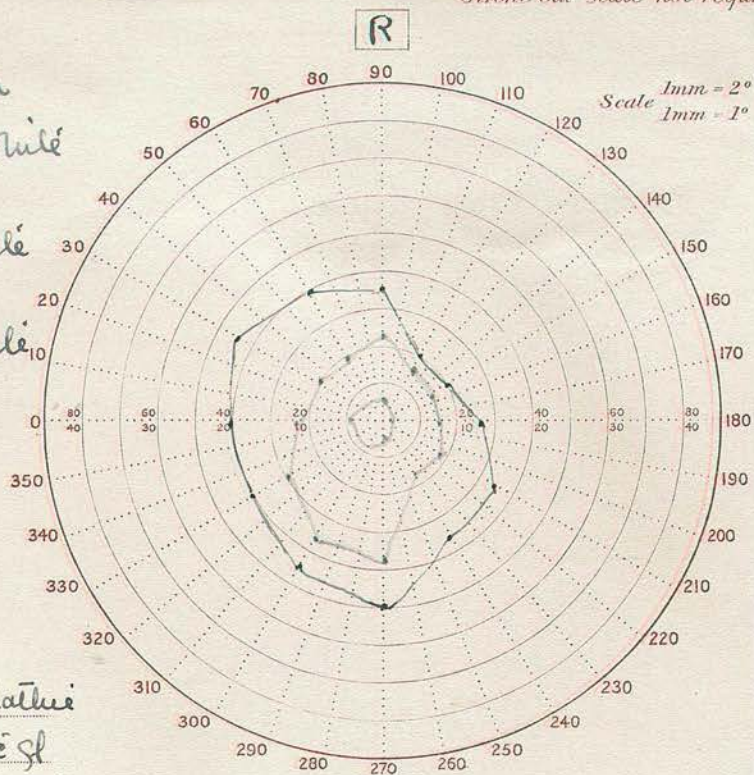
The case of Mrs Strathie which follows presents a very typical picture of the history and progress of a case of pituitary tumour. In this case one

eye was totally blind with atrophic optic nerve before being seen. This is an extreme illustration of the fact that the two sides are always involved at different stages. The remaining eye shows characteristic field changes. Notice particularly the presence of a central scotoma for colour. A 10/2000 red test object was not recognised at all in the upper temporal quadrant. In the lower temporal quadrant, it was recognised as white, in the lower nasal as an indefinite yellowish red, while only in the upper nasal quadrant was the brilliant red colour recognised as such.

MRS STRATHIE

Stroke out Scale not requir

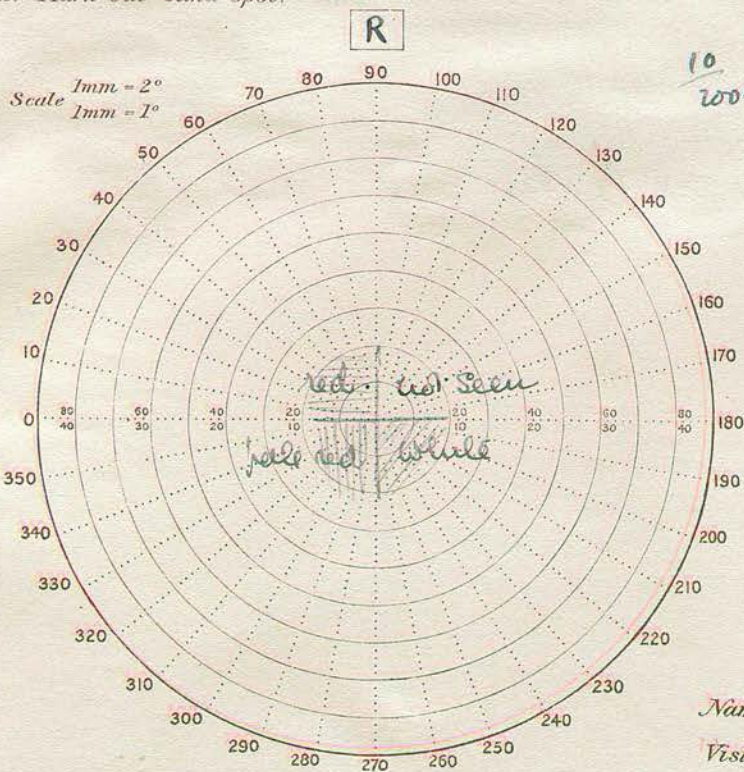
Fields for
10/330 white
4/330 white
1/330 white



Name Mrs Strathie
Vision 4/12 csl
Date 23.3.29.

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

d. Mark out blind spot.



10 red.
1000.

Name Mrs Strathie
Vision 4/12 csl
Date 23/3/29.

Case of Pituitary Tumour showing
Central Scotoma for Colour.

Mrs Strathie, 36 Blackie Road. Age 52.

15.3.29. Complaint: Sudden failure of sight of right eye and difficulty with reading, owing to inability to follow along the line of print to the right.

History: In 1916 suddenly noticed that sight of left eye had failed. At this time, following the birth of her youngest child, she ceased menstruating. During these years she has also suffered from severe headaches, which have been gradually getting worse. At no time, however, has there been any sickness. Teeth have been removed for the headaches.

Examination: Right pupil reacts directly to light, but not consensually.

Left pupil does not react to light directly, but does so consensually.

$$\text{R.V. } \bar{c} \quad \frac{+1.5}{+3} = 6/12. \quad 90^\circ$$

L.V. = No P.L.

$$\bar{c} \quad \frac{+4}{+3} = J 1 \quad 90^\circ$$

R. Fundus. Margin of optic disc appears blurred above.

MRS STRATHIE after operation

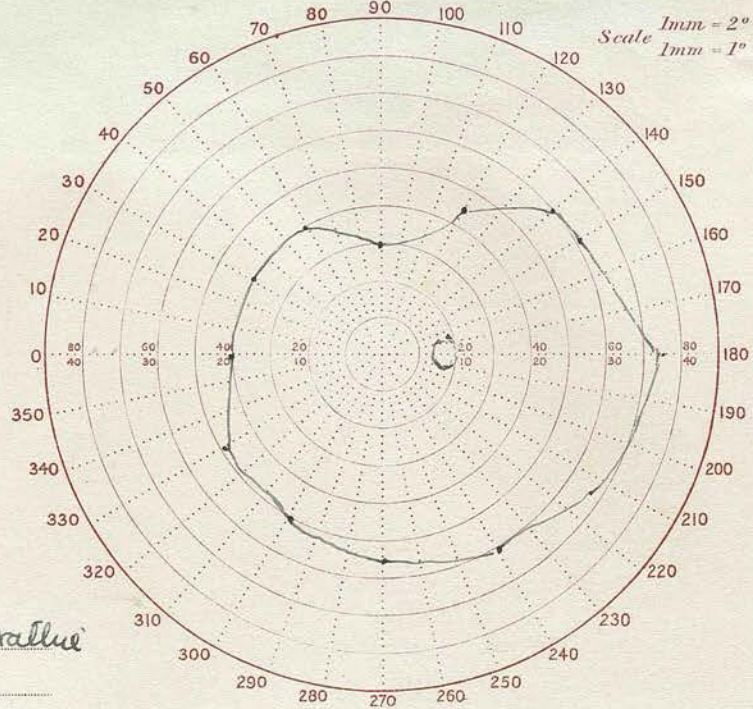
after operation

Stroke out Scale not required

1/330

R

Scale 1mm = 2°
1mm = 1°



Name Mrs Strathie

Vision 6/9

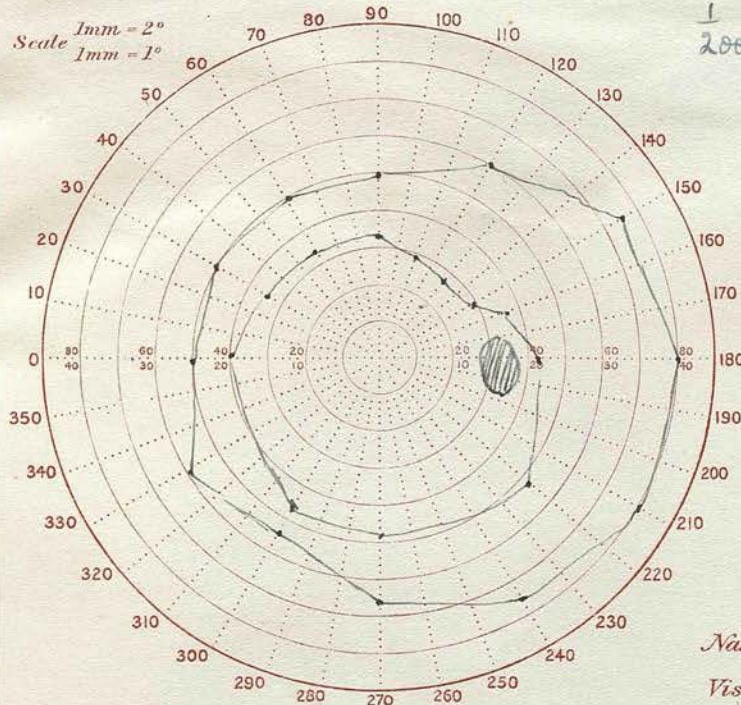
Date 6/5/29

TRAQUAIR'S CHART FOR PERIMETER OR BJERRUM SCREEN.

d. Mark out blind spot.

R

Scale 1mm = 2°
1mm = 1°



1/330 white

2000 white

138

20

2000

Name Mrs Strathie

Vision 6/9 c 8

Date 27/9/34

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L. Fundus. Condition of primary optic atrophy white disc with small vessels.

X-ray examination showed sella turcica to be enlarged and eroded behind.

23.3.29. Field examination at this date gave the following findings:-

Periphery examined on perimeter with 1/330 white object showed general contraction and a hemianopia to temporal side.

With 10/330 white, a well marked peripheral indentation in the upper temporal quadrant was present.

Screen Test. A 10/2000 red was not seen at all in the upper temporal quadrant.

In the lower temporal quadrant, was seen but called white.

In the lower nasal quadrant, was called pale red.

In the upper nasal quadrant, was recognised as pure red.

29.3.29. Seen by Mr Dott. Diagnosis, pituitary adenoma. Admitted to Hospital for operation.

10.4.29. Operation performed and a large amount of tumour material removed.

Pathological Report: Chromophobe Adenoma of Pituitary of compact cellular type, with very little supporting tissue.

6.5.29. Seen after operation.

$$\text{R.V. } \bar{c} \frac{+1}{+3} = 6/9$$

90°

$$\text{R.V. } \bar{c} \frac{+4}{+3} = \text{J 1 partly}$$

90°

Field when taken with 1/330 white showed a full periphery.

27.9.29.

$$\text{R.V. } \bar{c} \frac{+1}{+3} = 6/9$$

90°

$$\bar{c} \frac{+4}{+3} = \text{J 1.}$$

90°

Field with 1/330 white showed a full periphery.

With 1/2000 white, still showed some indentation in the upper temporal quadrant.

Central field for 10/2000 red was restored.

Clinical Significance of the Scotoma.

The macular fibres from each eye lie and cross in the posterior part of the chiasma. Therefore any lesion which involves this part of the chiasma will produce a central scotoma. It is unlikely that any lesion will involve the macular fibres only, so that the scotoma will be accompanied usually by peripheral defects. The scotoma, also owing to the arrangement of the macular fibres, will be bilateral, although in most cases at different stages. It is possible in very early stages that there is a unilateral scotoma. Obviously the more the posterior part of the chiasma is involved, the more extensive and pronounced will be the scotoma.

Scotomata are found in cases of an inflammatory nature and in rapidly increasing pressure. They are not present in slowly growing neoplasms. Therefore they seem to be an indication of activity. Great variety of size, shape and density are found, corresponding no doubt to the variations and fluctuations in different growths and in the same growth at different times.

Although tumour, i.e. pressure, is the commonest cause of scotomata, it must not be forgotten that other conditions also produce similar findings.

Retrobulbar neuritis occurs in the chiasma, though

much less commonly than in the nerve. Vascular lesions and syphilitic basal meningitis also occur, and all these conditions can produce similar scotomatous changes to tumour growth.

As regards diagnosis, the majority of tumour cases produce other well defined signs and chiefly show involvement of the sella turcica in X-ray pictures. In the dubious cases, time will show no improvement of symptoms and progressive growth of field defects.

GROUP IV.THE SUPRACHIASMAL PATHWAY.

The suprachiasmal pathway consists of

- (1) The optic tracts
- (2) The geniculo-calcarine pathway.

(1) The optic tracts are not commonly affected by disease, but when so, the lesions tend to resemble the chiasma, the commonest being tumour growth, syphilis and disseminated sclerosis.

The typical field defect produced by a lesion of one tract is an opposite sided homonymous hemianopia.

If the lesion is in the tract, that is, below the geniculate body, the hemianopia may show incongruity, a point which definitely points to a sub-geniculate lesion.

"The presence of a central hemianopic scotoma in homonymous hemianopia, otherwise suggestive of tract lesion, supports this diagnosis, as such scotomata are very uncommon in supra geniculate hemianopia." Traquair.

These central hemianopic scotomata may be produced by the lesion of disseminated sclerosis or by tumour growth, and as said before, resemble lesions of the chiasma.

(2) Geniculo-calcarine Pathway. The most common lesion affecting the suprageniculate pathway is undoubtedly vascular. Pressure is second but very rare, while toxic and inflammatory conditions and injuries (apart from war) practically do not occur.

The typical field defect produced by a lesion of this part of the visual path is a congruous hemianopia.- Compare this with the incongruity of the subgeniculate hemianopia.

The macular fibres have their ending in the cortex at the posterior part of the occipital centre and the foveal fibres at the tip of the occipital pole.

Minute central scotomata sometimes appear suddenly and are probably due to a vascular lesion of a small blood vessel in the cortex supplying the area in which the macular and foveal fibres end.

In ordinary civilian life, injuries of the occipital visual centre are rare, but the Great War provided a large number of such cases. Gordon Holmes has reported a case which suffered an injury

to the tip of one occipital lobe and showed an opposite sided hemianopic central scotomata. Other similar cases are known. (Sinclair).

SUMMARY.

The object of this Thesis is to demonstrate the value of the examination of the central field of vision as an aid to diagnosis. By picking out one particular sign, i.e. central scotoma, for study, it is not meant that by this sign alone can a diagnosis infallibly be made. It is never possible to make a diagnosis from any one sign or one method of examination. Rather must the entire clinical picture be taken into consideration. This picture is made up of innumerable small portions which, when fitted together like the parts of a jig saw puzzle, present to the examiner's view an entire and recognisable picture. In medical diagnosis, however, each single fact must be subjected to the minutest scrutiny and it is for the scrutiny of the part called "Central Scotoma" that this Thesis has been conceived and compiled.

In this summary there will be unavoidably a certain amount of repetition, as a short summary has been appended to the end of most of the divisions in order to clinch the outstanding points while still fresh in the reader's mind. Again, unavoidably a few important if rare conditions are omitted and

others remain unillustrated by original cases, as it has been thought better to give a description of the conditions only than to illustrate with cases taken from the work of other observers.

The subject has been divided under several headings, and the salient points of each will now be considered.

I. CHOROID AND RETINA.

Examination of the central field of vision in lesions of the choroid and retina is not of vital importance. In these cases diagnosis is made by examination with the ophthalmoscope. The only cases in which perimetry and campimetry will be found useful are those in which the visual loss does not correspond to the lesion, or in which no lesion can be seen ophthalmoscopically, i.e. certain cases of eclipse scotoma.

In some cases of obstruction of the retinal artery, whether by embolism, spasm, etc., central scotomata are found (De Schweinitz). The presence or absence of such a sign is not of importance in the diagnosis.

II. OPTIC NERVE.

By far the commonest part of the visual pathway

to be affected by conditions producing a central scotoma is the optic nerve. Among these conditions two stand out prominently, namely, Tobacco Amblyopia and Retrobulbar Neuritis. When dealing with conditions affecting the optic nerve, perimetry and scotometry are of incalculable value.

TOBACCO AMBLYOPIA is the commonest example of a group of cases which can be considered together under the heading Toxic Amblyopia.

In these cases which show no refractive error or no improvement of vision when rendered emmetropic, clear media and normal fundi, examination of the field of vision is of great service. The characters of the scotomata which are centrocaecal in position will be found described under the heading Tobacco Amblyopia in Group II. If all these characters are taken into consideration, and particularly if the scotomata are regarded in their relationship to the whole picture, no misdiagnosis should be made. The prognosis as to recovery of vision in these cases is good.

ALCOHOL AMBLYOPIA. Centrocaecal defects similar to those found in tobacco amblyopia are attributed by some to the use of alcohol alone or combined with tobacco. In Britain such changes have rarely if ever been found in a case of alcohol apart

from smoking, and are therefore considered more likely to be due to the tobacco.

Changes in the fields due to alcohol are to be attributed to the impurities therein and are of two main varieties:-

Acute, in which the field changes are pericentral rather than centrocaecal, and are very dense.

Chronic, which also show a pericentral scotoma with great loss of colour vision and poor recovery.

The Prognosis in amblyopia due to the impurities of alcohol is bad.

LEAD AND CARBONBISULPHIDE. Cases of poisoning by these substances which affect the vision are rare but show bilateral and pericentral scotomata.

RETROBULBAR NEURITIS.

ACUTE RETROBULBAR NEURITIS. Cases of retrobulbar neuritis resemble those of tobacco amblyopia in that no error of refraction, lesions of media or fundi are found. Hence again the importance of examination of the field of vision. Immediately a different picture is brought to view. The scotoma of retrobulbar neuritis is pericentral and commonly large and dense. If the scotoma is central but small, a careful ophthalmoscopic examination of the macular area should take place. The field changes increase rapidly in size and density in the early

stages, while resolution is slower and by means of retracement of the defect.

DISSEMINATED SCLEROSIS is a very common cause of retrobulbar neuritis, but it is not possible to diagnose the condition from the characters of the scotomata.

NASAL ACCESSORY SINUS Disease, on the other hand, is a rare cause of retrobulbar neuritis, but in it the scotomata are more persistent and fluctuate, which facts lead one to suspect the nasal sinuses.

CHRONIC RETROBULBAR NEURITIS. This is a rare condition and is more commonly bilateral than the acute type. The field changes are, however, similar.

TOTAL TRANSVERSE ACUTE RETROBULBAR NEURITIS is a very acute condition indeed. In fact complete blindness comes on with startling rapidity. Obviously the field examination is not of great value but often in the stage of recovery, if any, the presence of central scotomata can be demonstrated.

LEBER'S DISEASE is a hereditary form of the disease which shows similar field findings.

III. CHIASMA.

In the chiasma the commonest lesion is that of pressure, usually produced by a pituitary tumour. In a certain number of cases the field changes include central hemianopic scotomata. The presence of scotomata in such cases indicates involvement of the posterior part of the chiasma where the macular fibres run. They also indicate activity of growth or an inflammatory lesion, being never found in slow growing neoplasm, e.g. acromegaly.

IV. SUPRACHIASMAL PATHWAY.

(1) Tract.

The typical field defect of a tract lesion is a homonymous hemianopia showing incongruity. If to this is added the presence of a central hemianopic scotoma, the diagnosis is clinched, as central scotomata are very rare above the geniculate body.

(2) Suprageniculate Pathway.

Central scotomata are very rare in lesions of the suprageniculate pathway. They are found, however, in the obvious lesions of occipital war injuries. Also they sometimes occur suddenly and without any other defect in the field. They are

then attributed to a small vascular lesion in the tip of the occipital pole.

In conclusion I should like to state my great indebtedness to the work and friendship of Dr H. M. Traquair.